A Dissertation on

''A STUDY OF PATHOPHYSIOLOGY, MANAGEMENT AND FACTORS INFLUENCING DIABETIC FOOT ULCERS AMONG DIABETIC PATIENTS''

Dissertation Submitted to

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Dissertation Topic : A STUDY OF PATHOPHYSIOLOGY, MANAGEMENT AND FACTORS INFLUENCING DIABETIC FOOT ULCER AMONG DIABETIC PATIENTS.

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Certified that this is the Bonafide Dissertation in **''A STUDY OF PATHOPHYSIOLOGY, MANAGEMENT AND FACTORS INFLUENCING DIABETIC FOOT ULCERS AMONG DIABETIC PATIENTS''** was a work done by **Dr.ARUNKUMAR.N.R.** and submitted in partial fulfillment of the requirements for the Degree of **M.S.General Surgery, Branch I** of The Tamil nadu Dr.M.G.R Medical University, Chennai.

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DECLARATION

I Solemnly declare that the Dissertation titled **''A STUDY OF PATHOPHYSIOLOGY, MANAGEMENT AND FACTORS INFLUENCING DIABETIC FOOT ULCERS AMONG DIABETIC PATIENTS''** was done by me at Coimbatore Medical College during the academic year July 2016 – June 2017 under the guidance of **Prof. Dr.V.Elango, M.S.** this Dissertation is submitted to the Tamilnadu Dr.M.G.R Medical University towards the fulfillment of the requirement for the award of **M.S. Degree in General Surgery (Branch).**

PLACE: **Dr.ARUNKUMAR.N.R**

DATE:

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And also my special thanks to **Dr Veena** for completing my study.

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CERTIFICATE – II

This is to certify that this dissertation work titled **''A STUDY OF PATHOPHYSIOLOGY, MANAGEMENT AND FACTORS INFLUENCING DIABETIC FOOT ULCERS AMONG DIABETIC PATIENTS''** of the candidate **DR.ARUNKUMAR.N.R** with registration Number **221511303** for the award of **M.S in the branch of General Surgery**,I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains 96 pages from introduction to conclusion and the result shows **0% (Zero)** percentage of plagiarism in the dissertation.

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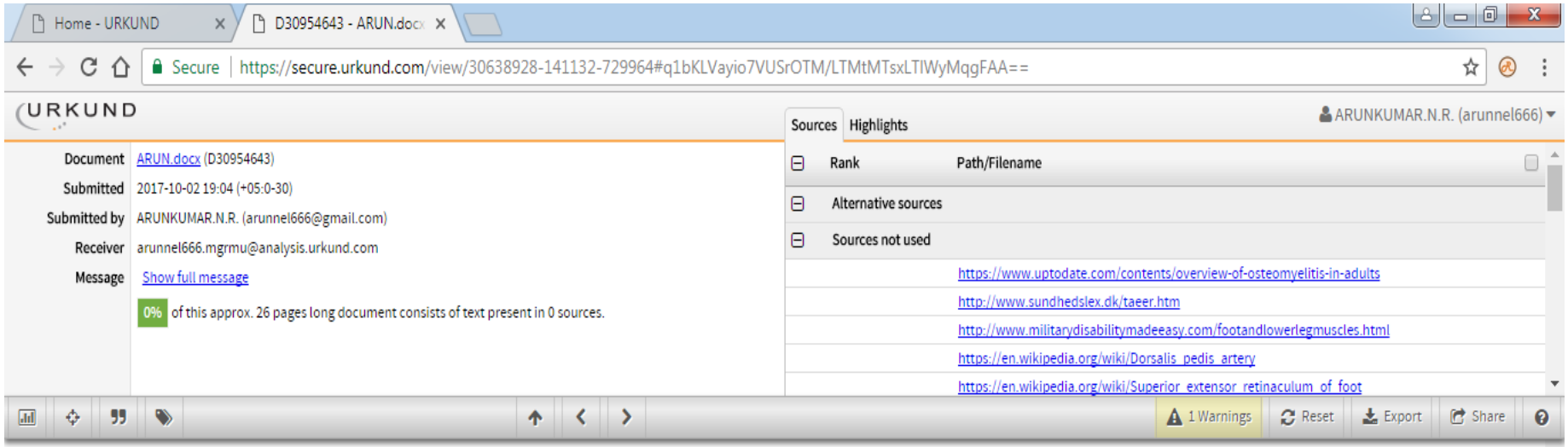
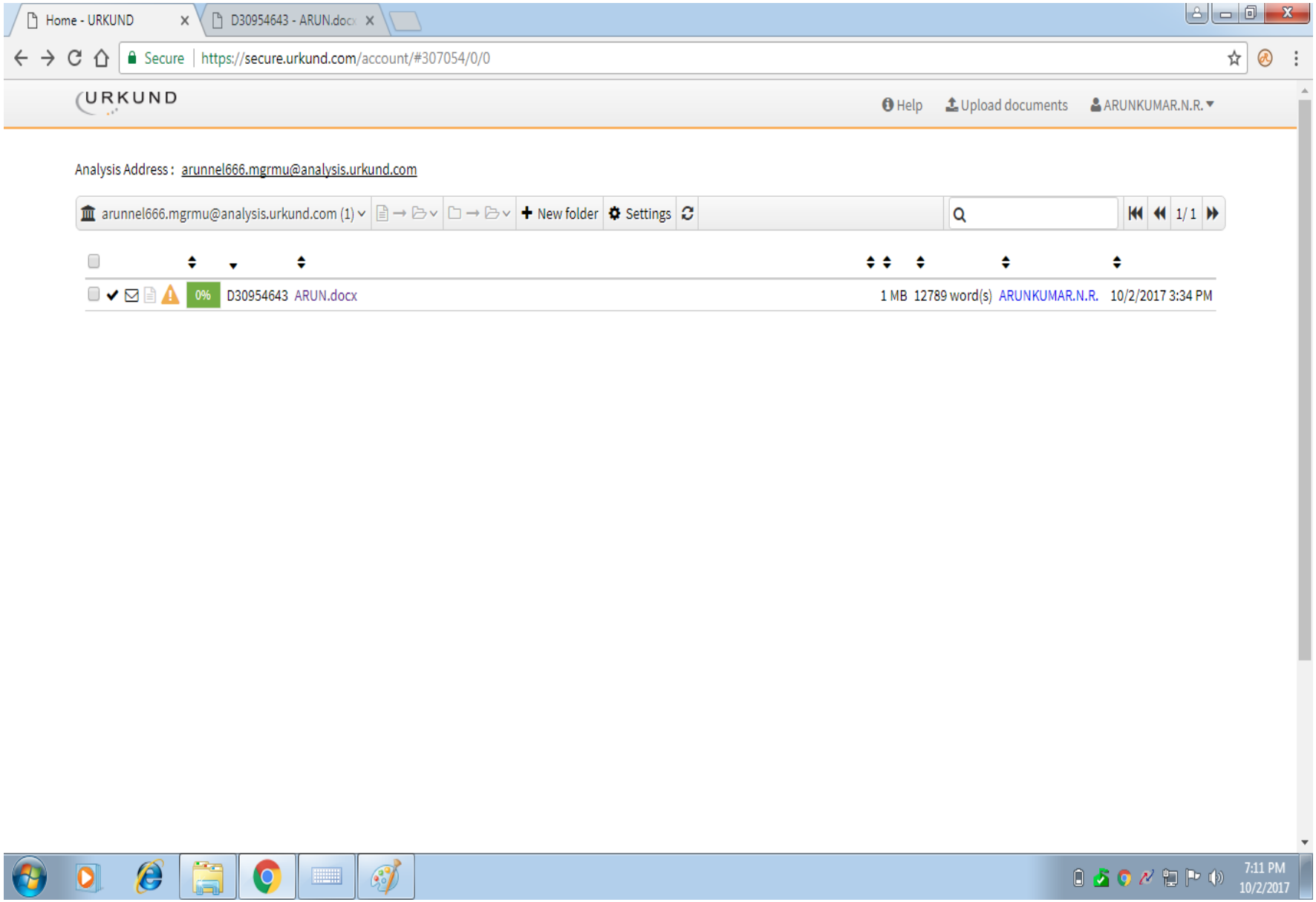


INTRODUCTION Diabetes is an endocrine disorder that has reached epidemic proportions worldwide. Overall 15 % of individuals with diabetes mellitus will have foot ulcer in their life time and the annual incidence of 2-5%. Diabetic foot is becoming a major concern of diabetic patients and those who treat them from quality of life, social and economic stand point. The word "Diabetic foot" means that the pathophysiological process of diabetes mellitus do something to foot, that puts at increased risk of tissue damage resulting to ulcer formation. (Payne & Florkowski, 1998). Foot damages such as ulceration, infection and gangrene are one of the important causes of hospital admission in patients with diabetic mellitus.(1) Natural history of diabetic foot. Evidence that the pathological process of Diabetes have put the foot for more risk for tissue damage has happened and foot is at risk for end stage complications (amputation). Of all complications of diabetes, those occurring in the foot are considered most avoidable. Epidemiology: (2) 3-5% of those with diabetes have a foot ulcer. 15% of all those with diabetes will, during their lifetime develops an ulcer. 1 4-5% of foot ulcers are increased by external trauma. Up to 20% undergo same side amputation within 12 months. Up to 50% undergo opposite side amputation within 1-3 yrs; 75% within 5 years. 3years increased death rate after complication is 20-50%. Most important risk factors are: • Loss of sensations. • Longer duration of diabetes. • High foot pressure. In 2000, The International Diabetic Federation endorsed the International Working on the Diabetic foot as a Consultative Section on the Diabetic foot. Organizations altogether established goals for the future of diabetic care worldwide (3). Goals • To inform the people about the diabetic foot problems worldwide. • To increase alertness of the diabetic foot among those at threat and those in place to act. • To influence healthcare decision makers that action is both achievable and reasonably priced. • To caution health care decision makers of the problems of not taking action. To inform people with diabetes of the actions they can avoid foot complications. 2 Multidisciplinary Management The plan in supervising diabetic foot is always to keep the patient at as low a phase possible. At each of the diabetic foot, it is essential to interfere early and to manage the foot to prevent further progression. No one person can manage the diabetic foot. Members of the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ABBREVIATIONS

CMCH	-	Coimbatore Medical College Hospital
IMV	-	Infectious Mononucleosis
HLA	-	Human Leukocyte Antigen
HDL	-	High Density Lipoprotein
LDL	-	Low Density Lipoprotein
DNA	-	Deoxyribonucleic Acid
DM	-	Diabetes Mellitus
IV	-	Intravenous
OGT	-	Oral Glucose Tolerance Test
GTT	-	Glucose Tolerance Test
CBP	-	Complete Blood Picture
SPP	-	Systemic Pulse Pressure
SVR	-	Systemic Venous Resistance
DKA	-	Diabetic Ketoacidosis
ABI	-	Ankle Brachial Index
VAC	-	Vacuum Assisted Closure
LA	-	Local Anaesthetic
AKA	-	Above Knee Amputation
BKA	-	Below Knee Amputation
SSG	-	Split Skin Graft
PUFA	-	Polyunsaturated Fatty Acid
I&D	-	Incision & Drainage
RBS	-	Random Blood Sugar
FBS	-	Fasting Blood Sugar
PPBS	-	Post prandial Blood Sugar

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INTRODUCTION

Diabetes is an endocrine disorder that has reached epidemic proportions worldwide. Overall 15 % of individuals with diabetes mellitus will have foot ulcer in their life time and the annual incidence of 2-5%. Diabetic foot is becoming a major concern of diabetic patients and those who treat them from quality of life, social and economic stand point.

The word “Diabetic foot” means that the pathophysiological process of diabetes mellitus do something to foot, that puts at increased risk of tissue damage resulting to ulcer formation. (Payne & Florkowski, 1998).

Foot damages such as ulceration, infection and gangrene are one of the important causes of hospital admission in patients with diabetic mellitus.(1)

Natural history of diabetic foot.

Evidence that the pathological process of Diabetes have put the foot for more risk for tissue damage has happened and foot is at risk for end stage complications (amputation). Of all complications of diabetes, those occurring in the foot are considered most avoidable.

Epidemiology: (2)

3-5% of those with diabetes have a foot ulcer.

15% of all those with diabetes will, during their lifetime develops an ulcer.

4-5% of foot ulcers are increased by external trauma.

Up to 20% undergo same side amputation within 12 months.

Up to 50% undergo opposite side amputation within 1-3 yrs; 75% within 5 years.

3years increased death rate after complication is 20-50%.

Most important risk factors are:

- Loss of sensations.
- Longer duration of diabetes.
- High foot pressure.

In 2000, **The International Diabetic Federation** endorsed the International Working on the Diabetic foot as a Consultative Section on the Diabetic foot. Organizations altogether established **goals for the future of diabetic care worldwide (3).**

Goals

- To inform the people about the diabetic foot problems worldwide.

- To increase alertness of the diabetic foot among those at threat and those in place to act.
- To influence healthcare decision makers that action is both achievable and reasonably priced.
- To caution health care decision makers of the problems of not taking action.

To inform people with diabetes of the actions they can avoid foot complications.

Multidisciplinary Management

The plan in supervising diabetic foot is always to keep the patient at as low a phase possible. At each of the diabetic foot, it is essential to interfere early and to manage the foot to prevent further progression. No one person can manage the diabetic foot. Members of the team will include physician, general surgeon, orthopedic surgeon, radiologist, expert nurse and podiatrist. (4)

It is helpful if the team works very much mutually, within the focal point of the diabetic foot clinic and also meets frequently to carry out ward rounds and x-rays conferences. Each team member should be available hastily in an emergency.

AIMS AND OBJECTIVES

AIM

1) The aim of this study is to study the current trends concerning the pathology, complications and treatment of diabetic foot ulcers.
2) To study the co-relation between atherosclerotic changes in the blood vessels of the lower limb & diabetic ulcers.
3) To study the bacterial flora & evolution of the ulcer with the relation to rigorousness of diabetes.

OBJECTIVES

1. PRIMARY

To assess the prevalence of diabetic foot ulcer and relative distribution according to age, sex, occupation and other factors among diabetic patients in patients attending CMCH.

2. SECONDARY

a. To study the mode of presentation and appearance of diabetic foot ulcers

b. To emphasize and enhance the knowledge of diabetic patients regarding self-care and regular diabetic foot evaluation.

c. To understand the pathology of diabetic foot ulcer and early recognition of complications of peripheral neuropathy and ischemia and using a multidisciplinary approach.

d. To avert the various complications due to diabetic foot ulcers and early management of its complications.

e. To study the different treatment modalities in management of diabetic foot ulcers.

REVIEW OF LITERATURE

There is considerable geographic variation in the incidence of both type 1 and type 2 Diabetes mellitus. The variability is likely due to genetic, behavioural, and environmental factor. Diabetes mellitus prevalence also varies among different ethnic populations within a given country (5).

Diabetes mellitus

Diabetes mellitus is the most common endocrine disorder 6 characterized by metabolic abnormalities and by long term complications involving eye, kidney, nerves and blood vessels.

W.H.O CLASSIFICATION OF DIABETES MELLITUS (6)

Primary

Type 1 – Insulin dependent diabetes mellitus (IDDM).

Type 2 – Non insulin dependent diabetes mellitus (NIDDM).

Secondary

- Diabetes caused by pancreatic disorder.
- Diabetes caused by hormonal abnormalities.
- Chemical induced diabetes mellitus.
- Diabetes caused by insulin receptor abnormalities.
- Diabetes associated with genetic syndromes.
- Diabetes of other causes.

ENVIRONMENTAL EVENT

The fact that monozygotic twin of IDDM patient may remain asymptomatic proves that there are other factors other than genome in the pathology of IDDM. Environmental factors also play a role. In many cases it is a viral infection of beta cells. Mumps, Hepatitis, IMN, Congenital Rubella. Coxsackie virus may trigger the disease. It is also postulated that exposure to cow s milk products early in life predispose to autoimmune diabetes. The proposed mechanism is bovine albumin acting through mechanism of molecular mimicry.

NIDDM is 7-8 times more common than IDDM (7).

PATHOGENESIS OF IDDM (8)

Pathology of IDDM is due to destruction of beta cells of pancreas. Pathogenesis starts with genetic susceptibility to disease, some environmental factors also play in the pathogenesis. Viral infection usually triggers the destruction. During pathogenesis the patient is initially non – diabetic. Progressively beta cells are infiltrated and destroyed by macrophages and T lymphocytes, this stage is called insulinitis or isleititis. During this period reserve of insulin progressively diminishes and when it is insufficient to maintain blood glucose level within normal the patient develops diabetes. Genetic susceptibility to

IDDM probably involves more than one gene. Candidate loci are proposed in chromosomes 2, 6, 11 and 15. It is HLA associated.

PATHOGENESIS OF NIDDM

Aetiopathogenesis include both insulin resistance and beta cell defects.

- Major environmental factor is obesity.
- Genetics: Maturity onset diabetes of the young shows autosomal dominant penitence. Other varieties of NIDDM are polygenic.

PATHOPHYSIOLOGY

Patient with Type 2 NIDDM have physiological defects. Abnormal insulin secretion and insulin resistance.

CLINICAL FEATURES OF DIABETES MELLITUS

IDDM: It usually begins below 40 years. Peak incidence is within 14 years. Onset of symptoms is often abrupt with thirst, excessive urination or increased appetite.

NIDDM: Usually begins in middle life or later. Patient is often obese. Symptoms begin gradually, frequently diagnosed in asymptomatic patient during blood sugar measurements. Plasma insulin level is normal to high but there is relative insulin deficiency in relation to high blood sugar.

General characteristics OF NIDDM AND IDDM are given below (9):

Characteristics	IDDM	NIDDM
General factors	Chromosome 6	Unknown
Age of onset	Below 40	More than 40
Body habitués	Normal or wasted	Obese
Plasma insulin	Low or absent	Normal or high
Acute complication	Ketoacidosis	Hyperosmolar coma
Insulin therapy	Responsive	Responsive to resistant
Response to Sulfonyl urea therapy	Unresponsive	Responsive

SURGICAL ANATOMY OF FOOT (10)

SKIN AND NAILS

The skin of dorsum of foot is thin and highly flexible, containing hair follicles; sweat glands and scanty sebaceous gland. Hairs are sparse and thin. It is less than 2mm thick and few fibrous septa penetrate to deeper facial structures. The plantar skin is 5mm thick especially over those points which bear weight viz. Heel, ball of big toe and lateral margins of the sole. It has no hair follicles or sebaceous glands but glands are numerous.

NERVES

Cutaneous nerves are arranged in the following way. The medial plantar nerve supplies the three and half digits on the big side of the foot. The lateral plantar nerve supplies one and half digits. The medial calcaneal branches of the posterior tibial nerve supply the skin under the heel.

The motor and sensory components of the sciatic nerve supply the foot. The innervations to the sole is from medial calcaneal branch of tibial nerve. The dorsum of the toes is supplied by the digital branches of these nerves except the saphenous on the terminal phalanges the supply is from the plantar nerves.

VASCULATURE OF FOOT

All the arterial supply of the foot is derived from popliteal artery. The anterior tibial artery enters the extensor compartment of the leg and becoming in the foot the dorsalis pedis artery. The posterior tibial artery divides into medial and lateral plantar arteries. The medial plantar artery runs forward on the medial side of the medial plantar nerve. The artery supplies medial side of foot and its digital supply is restricted practically to the big toe. Lateral plantar artery crosses the sole obliquely on the marginal side of the nerve, just deep to the first layer of the sole

towards the base of the fifth metatarsal bone. The plantar arch curves convexly forward across the base of fourth, third and second metatarsals and is joined in the proximal part of the first inter-metatarsal space by the dorsalis pedis artery from the convexity of the plantar arch, plantar metatarsal arteries run forward and bifurcate to supply the four web spaces and digits. The veins accompaning perforating arteries take most of the blood from the sole and from the interosseous muscles to the dorsal venous arch.

MUSCLES OF THE FOOT

The muscles in the extensor group are located anteriorly in the leg, they include tibialis anterior, extensor hallucis longus, laterally are the peroneal muscles. The flexors are in the posterior compartment of the leg. The deep fascia encloses the muscles in the leg. The gastrocnemus arises on the distal posterior femur. In the sole of the foot the plantar aponeurosis is the most superficial layer, the fibers of plantar fascia is divided into five processes beneath fascia the fascia. The muscles in the sole of the foot are categorized into four layers only the muscles of first layer cover the whole extent of the foot muscles in the first layer include flexor digitorum brevis, abductor hallucis and abductor digiti minimi.

In the second layer are tendon of flexor hallucis longus, flexor digitorum accessories (quadrates plantae) and the lumbricals. In the third

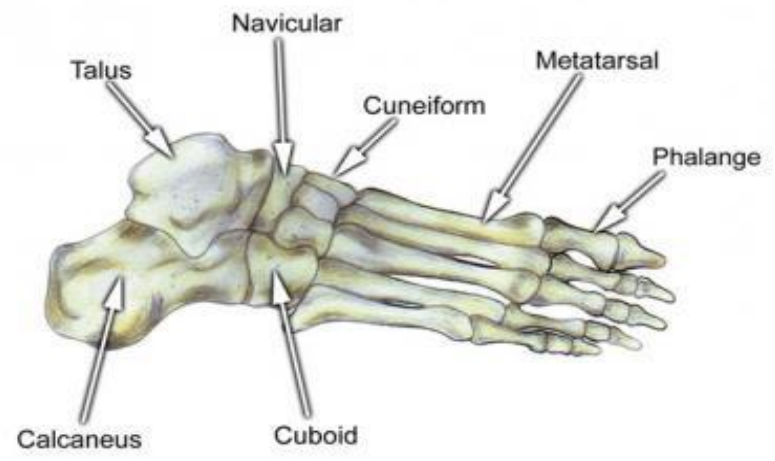
layer are flexor hallucis brevis, abductor hallucis and flexor digiti minimum brevis. In the fourth layer is peroneous longus tendon, of the tibialis posterior, four dorsal interossei and three plantar interossei.

PERIOSTEUM

Periosteum is a fibrous membrane investing the bones except at their articular surfaces. It is adherent to the bone and varies in different places. In adult bone it is firmly adherent and especially so at the insertion of tendons and ligaments where more periosteal, fibers penetrate into bone as the perforating fibers of Sharpey.

Periosteum consists of two layers. The outer layer is composed of coarse fibrous connective tissue containing few cells but as numerous blood vessels and nerves. The inner layer is less vascular but more cellular and contain many elastic fibers.

BONES OF THE FOOT



The bones of the foot are the tarsal bones, metatarsals and the phalanges. The tarsal bones are the calcaneum, the talus, the navicular, the cuboid and the three cuneiform bones. Calcaneum is the largest bone of the foot and forms the prominence of the heel it articulates with talus above and cuboid in front. Talus carries the whole body weight. It lies on weight bearing calcaneum below the tibia and communicates thrust from one to the other. Navicular bone can be seen and felt on the medial border of the foot. Cuboid bone is rather wedge shaped narrowest at the lateral margin and broadest medially, where it articulates with lateral cuneiform.

Cuneiform bones, all three are wedge shaped. Medial is largest and the edge lies upwards. Intermediate is smallest. All the three articulates posteriorly with navicular and anteriorly with metatarsal bones. This completes medial longitudinal arch.

METATARSAL BONES AND PHALANGES

The metatarsal bones and phalanges resemble metacarpals and phalanges of hand. Each has a head distally, a shaft and a base proximally. There are five metatarsals and they are numbered from medial to lateral side. The first metatarsal bone is large and strong and it plays an important role in supporting the weight of the body. Second to fifth metatarsals have slender shafts. Each toe has three phalanges except the big toe, which posses only two.

PATHOPHYSIOLOGY OF DIABETIC FOOT

It has been recognized that persons with diabetes are prone to foot problems.

Recent advances in molecular biology have added substantial insight into the pathophysiology of the disease and opened new avenues for treatment (11).

The predisposing factors to pathologic changes in the foot of a diabetic are:

1. Metabolic factors – hyperglycemia
2. Vascular changes
3. Neuropathy
4. Infection

Polyol pathway.

Glucose → sorbitol → accumulation in nerves, retina, kidney.

Hyperglycemia results in increased levels of sorbitol in the cell, which acts like an osmolyte a competitive inhibitor of myoinositol uptake. This preferential shunting of glucose through the sorbitol pathway results in decreased mitochondrial pyruvate utilization and decreased energy production. This process is termed hyperglycemia induced pseudohypoxia.

Glucose + protein amino group

↓

Early glycosylation products (poorly irreversible)

↓

Advanced glycosylation products (completely irreversible)

↓ ↓ ↓

Endothelium Macrophages Extracellular matrix protein

↑Procoagulant ↑ chemotaxis ↑ cross linking of collagen

Activity GF synthesis Trapping of serum proteins

↑Permeability Monokinins Susceptibility to enzymatic

Secretion degradation

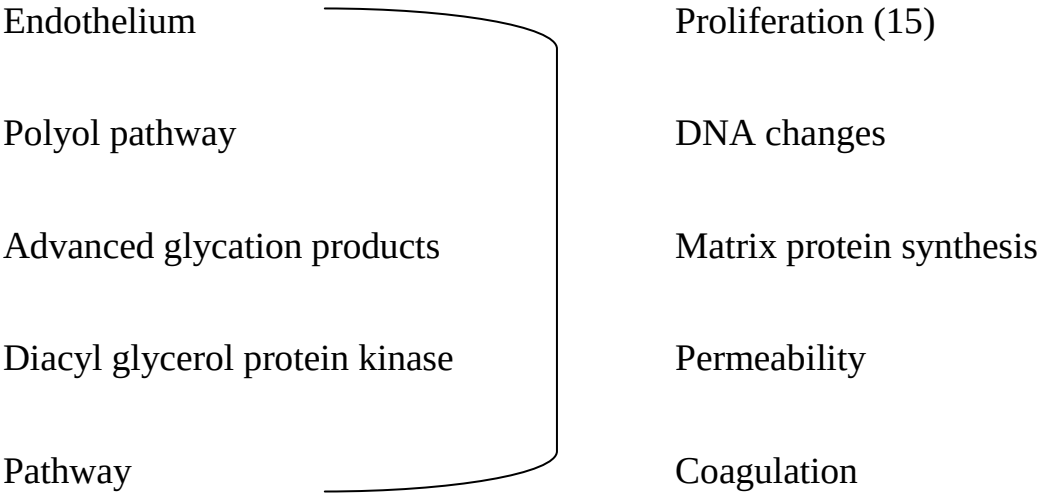
↑Activation of NF-KB

Risk factors: (12)

- Hypertriglyceridemia (VLDL)
- Low levels of HDL
- Increase in cholesterol : lecithin ratio

Pathogenesis : Enhanced non – enzymatic glycosylation of lipoprotein has been shown to impair the binding of glycosylated LDL to the LDL receptor (13). Glycosylated LDL enhances the formation of cholesteryl ester and accumulation human macrophages – formation of foam cells characteristic of early atheromatous lesion (14).

It is also noted that, vascular smooth muscle cells exhibit increased growth on exposure to high glucose in vitro.



Microangiopathy:

Hyperglycemia causes thickening of basement membrane of small vessels and capillaries due to incorporation of carbohydrates into basement membrane by induction of enzymes such as glycosyl, gactosyl transferase. The chemical changes in the basement membrane are:

- Increased hydroxylysine and glucose disaccharide content
- Decrease in proteoglycan and heparin sulfate

- Increase in collagen type 4
- Decrease in lysine
- Decrease in laminin

Thickening interferes with transfer of oxygen and nutrients to the tissues migration of leucocytes to area of sepsis, there by delaying wound healing (16).

Hematological Changes:

The haemotological abnormalities are increased plasma and blood viscosity such as alteration in the plasma protein profile and disturbance in erythrocyte behavior. Erythrocytes are prone to increased aggregation and also showed reduced deformability (17).

Haemostatic imbalances originate from acquired coagulation defects. The abnormalities of haemostatic system in DM are:

Endothelium - ↓ Prostacyclin

Platelets - ↓ Tissue factor production

↓ Hypersensitivity to agonists

↓ Aggregation

↓ Membrane fluidity

↓ Platelet volume

Coagulation abnormalities are :

Coagulation factors :

↑ Fibrinogen

↑ Factor 7 and 8

↑ Von willebrand factor

Coagulation inhibitors:

↓ Antithrombin III activity

↓ Heparin cofactor II activity

↓ Thrombin – anti thrombin complex levels

↓Protein C levels

Fibrinolysis abnormalities:

↑ Plasminogen activator inhibitor

Megakaryocyte platelet system is activated in diabetes mellitus.

Gangrene

Type I- Patchy gangrene

Type II- Extensive gangrene

Gangrene Foot

Neuropathy in the diabetic foot

Peripheral neuropathies are found in 55% of diabetes. The incidence of neuropathies increases with duration of disease and episodes of neuropathies increases with duration of disease and episodes of hyperglycemia. Peripheral neuropathy clearly renders the patient to unrecognized injury, which potentiates the risk of bacterial invasion and infection (18).

DEFINITION OF DIABETIC NEUROPATHY: Is controversial, however the presently accepted definitions is demonstrable (clinical or sub clinical) disorder of somatic or autonomic parts of peripheral nervous system occurring in patients with DM (19).

Signs & Symptoms- Paraesthesia, Hyperaesthesia, Hypoaesthesia, Radicular pain, Loss of deep tendon reflexes, Loss of vibratory and position sensation, Anhydrosis, Heavy callus formation over pressure points, Infection complication of trophic ulcers, Foot drop, changes in bones and joints (20).

Radiographic changes- Demineralization, Osteolysis, Charcot joint

Staging system (21)

Stage 0 : No neuropathy (no symptoms and fewer than 2 abnormalities on testing)

Stage 1 : No symptoms, but 2 or more abnormalities of functional testing

Stage 2 : Symptoms of lesser degree than stage III along with 2 or more functional abnormalities

Stage 3 : (Disabling neuropathy) Disabling symptoms and 2 or more functional abnormalities.

The functional tests done are- Nerve conduction, Neurologic examination, Quantitative nerve testing of muscle strength, Threshold of vibratory ,cooling or warming sensation and Autonomic function.

CLASSIFICATION:

1) Symmetrical polyneuropathies

- Sensory or sensimotor polyneuropathy
- Symmetrical proximal lower limb motor neuropathy
- Acute or sub acute distal motor neuropathy
- Autonomic neuropathy

2) Focal or multifocal neuropathies

- Cranial neuropathy
- Traumatic and limb mononeuropathy
- Asymmetrical lower limb motor neuropathy
- Mixed forms

Risk factors- Duration of diabetes and neuropathy**,** Male and tall individuals**,** Elderly diabetics**,** Excessive consumption of alcohol in diabetes**,** Smoking tobacco and Diabetes with lower limb ischemia caused by peripheral vascular disease.

Treatment of diabetic neuropathy:

a) Aldose reductase inhibitors

- Glucose aldose sorbitol
- Reductase

Drugs used are:- Alrestatin**,** Epalrestal**,** Sorbinil**,** Tolrestate**,** Ponatrestal.

b) Gangliosides: results in some degree of recovery following nerve injury by stimulating sprouting mechanisms, activation of Na+ - K+ ATPase and promotion of production of nerve growth factors by Schwann cells.(22)

c) The attainment and maintenance of normal blood glucose & lipids, in conjugation with ideal body weight remain the corner stone of improvement of DM.

Symptomatic treatment for painful diabetic neuropathy. Simple reassurance is the best treatment. Simple analgesics for mild cases

Drugs used are-

a. Tricyclic antidepressents - Amitryptaline, Despiramine, Fluphenazine
b. Anticonvulsants – Carbamazepine and Phenytoin
c. Baclofen
d. Clonidine
e. Lidocaine, Mexiletine
f. Transcutaneous electrical nerve stimulator

Infections: The patients with DM are prone to infection than normal individual. In a normal individual the flora of the lower leg and foot are restricted because of following reasons:

1. Skin temperature is much lower than optimum for many human pathogens
2. Metabolic products of skin have antimicrobial chemical effect
3. Acid surface of dorsum of foot and lower leg, making cervical dependent on the ability of various microbes to resist drying.
4. Thick stratum corneum.

Of all the infections seen in diabetic patient, bacterial and fungal infections of the skin are most common.

Predisposing factors- Vascular deficiency, Neuropathy, Resistance to infection could be due to leucocyte mobilization, Defective chemotaxis, Neutrophil bactericidal defects, Defect in formation of reactive oxygen metabolites (23)

Arterial insufficiency locally tissue pressure& metabolism

↓ ↓

Increased tissue demand for oxygen

↓

Increased extra vascular tension

↓

Local production of tissue destructive enzymes

↓

Local thrombosis and small vessel occlusion

↓

INFECTION

Common organisms are: aerobes/anaerobes

Aerobes:

1. Gram negative bacilli - P.mirabilis, E.coli, P. Aeroginosa, E.aerogenes
2. Gram positive bacilli - Enterococcus, S.Aureus, Group B streptococcus

Anaerobes:

1. Gram negative bacilli - B.fragilis, B.ovatus, B.ureolyticus
2. Gram positive bacilli - P.magnus, P.anaerobes, C.bifirmentans

The infections are polymicrobial in DM.

Laboratory Diagnosis:

Gram stains, aerobic & anaerobic bacterial culture should be obtained from all suspected foot infections. Materials collected by curettage of the case of the ulcer are best specimens.

Anaerobic samples should be taken using a syringe, injecting the sample through the diaphragm in the tube cap and transported in oxygen free medium.

Treatment:

Initial broad spectrum I.V antibiotics coverage for all of these bacteria is indicated on admission. Appropriate changes are made according to bacterial sensitivity studies.

Commonly used antimicrobial agents are- Ciprofloxacin, Ampicillin clavulanate, Ampicillin sulbactum, Ceftazidime, Ceftriaxone, Cefoperazone, Piperacillin tazobactum, Imipenam cilastatin, Clindamycin and Metronidazole.

Aggressive nutrition support, Complete rest of injured part, Extensive debridement, adequate dependent drainage, appropriate arterial reconstruction and well chosen, conservative amputation are mandatory.

Development and complication of diabetic foot ulcers:

Neuropathy and ischemia are the 2 important predisposing factors for the formation of diabetic ulcer. Physical or mechanical stress is required for an ulcer to develop.

Predisposing factors :

1. Motor neuropathy: motor involvement results in weakness of intrinsic muscles of foot. This causes imbalance between long flexors and extensor tendons, which results in typical cavus or high arched foot along with clawing of toes. These factors lead to

increase in pressure under metatarsal heads & heal, ultimately leading to ulceration (1).

2. Sensory neuropathy: The major factor in neuropathy is loss of protective pain sensation and repetitive injuries to an insensitive extremity.
3. Autonomic neuropathy: cause central complications such as postural hypotension.

Effect on neuropathy on circulation:

Loss of sympathetic tone, Peripheral flow, loss of postural vasoconstriction, Pressure in capillaries, Basement membrane thickening, AV shunting.

Sensory component is assessed by "Bio Thesiometer". This measures vibration perception threshold.

The readings are taken at medial malleous & pulp of the great toe to produce mean score. Inability to feel vibration at 35 V or more is indicative of peripheral neuropathy.

This can also be studied by Semmes – Weinstein hairs which consists of nylon filaments of equal length but different diameters, that buckle at a constant force.

EXAMINATION OF FOOT IN DIABETIC PATIENTS

	Clinical examination	Objective testing
Shape & deformities	Toe deformities, prominent metatarsal heads, hallux valgus, callus, Charcot's deformity	Radiograph of foot Foot pressure studies
Sensory function	Vibration, thermal proprioception, Semmes Weinstein filament	Biothesiometry thermal threshold testing
Motor function	Wasting weakness Ankle reflexes	Electrophysiological tests
Autonomic function	Reduced sweating, callus, warm foot, distended dorsal foot veins	Quantitative sweat test Thermograph of skin temperature
Vascular status	Foot pulses, pallor cold feet, oedema	Non- invasive Doppler studies TcPO2

Ischemia : These ulcers are relatively uncommon. Common over 1st and 5th metatarsal heads. In the lower limb, vessels most commonly affected are the distal superficial femoral, tibial and peroneal arteries.

Initiation of ulceration:

Ischemic ulcers are developed by physical or mechanical stress.

Wagner diabetic foot lesion grading system.

Wagner (1983) grades lesions of diabetic foot from 0-5 by depth and extent.

Grade	Description
O	No ulcer but high risk foot
1	Superficial ulcer (commonest site is head of 1st Metatarsal)
2	Deep ulcer with no bony involvement
3	Abscess with bony involvement
4	Localised gangrene
5	Gangrene of whole foot

Liverpool classification system

Classification	Description
Primary	Neuropathic Ischemic Combined
Secondary	Uncomplicated & Complicated

Complications in diabetic ulcers- Cellulitis, Abscess, Osteomyelitis, Septicemia, Necrosis, Charcot foot.

DIABETIC FOOT COMPLICATIONS

Osteomyelitis is a common problem in the diabetic foot. This compromises blood supply and decreased sensation in the diabetic foot makes diagnosis and treatment of osteomyelitis difficult.

Stage 1 - It is a simple infection with no permanent anatomic damage. This is medullary osteomyelitis in a bone, acute septic arthritis of a joint or cellulitis of soft tissue.

Stage 2 - Is superficial periosteal or cortical osteomyelitis, chondrolysis, sub acute septic remains arthritis, or ulcerated soft tissue.

Stage 3 - Infection is deeper but remains localized. It involves both the cortex and medullary canal for osteomyelitis, bone about the the joint for septic arthritis, or an abscess in soft tissue.

Stage 4 - Diffuse infection, diffuse osteomyelitis (nonunion), end stage septic arthritis (unstable point), or a permeating necrotizing infection (gas gangrene, necrotizing fasciitis).

The UTMB Staging system for Musculoskeletal infections

Anatomic type	Bone OM	Septic arthritis	Soft tissue Cellulitis
I Simple	Medullary OM	Simple SA	Cellulitis
II Superficial	Superficial OM	SA with chondrolysis	Ulcer
III Localized	Local OM	SA with localized OM	Abscess
IV Diffused	Diffuse OM	Unstable joint	Permeative

Physiologic class

A Host Good immune system and delivery

B Host Compromised locally (B1) or Systematically (Bs)

C Host Requires suppressive or no treatment, minimal

Disability, treatment worse than disease; not a Surgical candidate

Clinical Stage

Type + class = Clinical stage

Example : Stage IV (Bs) Osteomyelitis = a diffuse lesion in a

Systematically compromised host.

Abbrevations : OM – Osteomyelitis, SA – Septic arthritis

Permeative soft tissue infections includes gangrene, necrotizing fascitis, etc

Causes for Charcot's joint

Charcot s joint is a type neurogenic arthropathy.

1. Diabetic neuropathy
2. Autonomic nervous system dysfunction
3. Sepsis of the part
4. Ischemia of the part

INVESTIGATIONS

The following investigations are to be done for the diagnosis and treatment of diabetic foot.

To demonstrate the extent and severity of the disease process.

To screen diabetic patients for peripheral vascular insufficiency.

To confirm and control the intercurrent diseases interfering with the healing process.

URINE EXAMINATION

ALBUMIN SUGAR- Microalbuminuria > 300mg/dl indicates future risk for Diabetic Nephropathy.

GLYCOSURIA : Glucose in urine in concentration less than 0.1% to be considered normal. Benedict s qualitative and quantitative test, enzymatic test, and clinical tests are used. The most specific method available is

glucose oxidase test which oxidase glucose to gluconic acid and liberates hydrogen peroxide, which is measured. This test applies either to blood or urine.

KETONURIA : If glucose is present in urine ketone bodies should also be determined can be detected by Rothera's acetone test. It is the first sign to be recognized in ketosis.

BLOOD EXAMINATION

FASTING BLOOD SUGAR : Hyperglycemia is most decisive indication of diabetes. It is estimated by folin Wu or Somgy's nelson method. Fasting blood sugar more than 120 mg% is indicative of diabetes.

POST PRANDIAL BLOOD SUGAR: After overnight fast, the patient is given breakfast of 100gm of carbohydrates or 100gm of glucose load then after venous blood is checked for glucose level every half hour for two hours, if it exceeds 180mg% is indicative of diabetes meliitus.

ORAL GLUCOSE TOLERANCE TEST: Sample of blood and urine are taken prior to the test 100gm of glucose in water are administered orally to an overnight fasting patient. Once again venous blood and urine samples are taken half hourly intervals. For about two to three hours blood samples are examined quantitatively and urine sample quantitatively for glucose. This gives glucose tolerance curve.

In normal subject fasting blood sugar is 80- 120 mg% and peak of the curve is not above 180gm%. The blood sugar value returns to normal fasting level or slightly lower at the end of two hours and there is no sugar in any sample.

INTRAVENOUS GLUCOSE TOLERANCE TEST: Intravenous GTT is indicated in certain conditions where there is adequate absorption of glucose from intestine as in steatorrhea, pancreatic islet cell tumors, Addison's disease, hypopituitary states or post gastrectomy syndrome.

A sterile glucose solution of 20 – 30 gm(20%w/v) over one to three minutes period in an amount of 0.5gm/kg bodyweight is administered intravenously.

Blood samples were taken thereafter and estimated for sugar. In normal individuals, the fasting level is regained in about 1 hour where as in diabetes; the blood glucose level remains high as in OGT.

CORTISONE GLUCOSE TOLERANCE TEST: This test may reveal prediabetic patients especially in relatives of known diabetics. Cortisone promotes intolerance in latent or mild diabetes. After performing a initial glucose tolerance test a standard dose of cortisone 50mg for adults is given parentarally eight and half hours and again two hours before a glucose tolerance test. A positive test shows a blood glucose

concentration of 140mg% or higher with 2 hour specimen. Follow up studies are necessary for such individuals.

In diabetes the blood glucose may rise to a high peak value of 300mg% or even more subsequently very slow fall sets in, so that may hours may relapses before the fasting blood sugar levels is recognized is regained and urine samples contain sugar. Cholesterol and triglycerides usually raised, can be assessed chemically or by electrophoresis.

BLOOD UREA:

Indicates the renal function but may vary with the hydration of the patient.

SERUM CREATININE

This is more sensitive indicator of the renal function.

COMPLETE BLOOD PICTURE:

It is essential to have it as a baseline investigation. It may provide information about concomitant anemia requiring correction by blood transfusion. Repeated assessment of CBP is important when uncontrolled infection is suspected, as leucocytosis is invariably present in such cases.

LIPID PROFILE:

These help in detecting the familial links in this disease and also reflected the severity of vascular disease which might be present.

BLEEDING AND CLOTTING TIME:

Not only as a routine, these investigations have specific relevance with regard to platelet function and response to the vessels and blood corpuscles. They may require correction when contemplating may surgery on the patient.

CULTURE AND SENSITIVITY TESTS:

Pus from infected area is cultured for microorganisms and their sensitivity to various antibiotics is tested so that appropriate antibiotic can be administered to control the infection.

X-RAY

X – ray of the foot should be taken if there is any suspicious infection deep to the foot, e.g: abscess or osteomyelitis. The sign, which suggests the presence of osteomyelitis, is destruction of bone commonly seen at metatarsophalangeal joint or in the interphalangeal joint of the great toe.

NON-INVASIVE EVALUATION

1) Toe pressure

They provide a highly accurate method for determining the success in the healing of an ulcer or in minor amputation. A toe pressure of 20-30 mmHg below which healing is doubtful.

2) Duplex scanning with ultrasound analysis (Doppler study)

The recorded Doppler signal is used in two ways:

- To measure segmental systolic pressure
- To provide flow velocity wave form pattern for analysis.

This combines B- mode anatomic capabilities of revealing the location and amount of vessel lumen and stenosis can be recorded with Doppler derived velocity recordings.

3) Others

PHOTOPLETHYSMOGRAPHY

SEGMENTAL PRESSURE

WAVEFORM EVALUATION

INVASIVE TECHNIQUES

1. Angiography

Percutaneous femoral angiography

Anatomic evaluation of the vascular supply to the leg and foot require arteriography. In young patients with vascular insufficiency diagnosis of obstruction can be made when arteriogram show severe diffuse atherosclerotic disease involving the tibial and peroneal arteries. The possibility of large vessel stenosis are occlusion superimposing on distal diabetic vascular disease is most important indication for angiography.

Complications- Local bleeding and hematoma, Thrombosis, Peripheral embolization, High doses of contrast will cause reduced renal function, Idiosyncratic or allergic reaction to contrast mainly Nausea, vomiting and itching, Hypertension, laryngeal edema and Bronchospasm

2. Digital subtraction angiography

The term digital subtraction angiography refers to visualization of vessels using digital fluoroscopic techniques for image enhancement.

Advantages:

Digital subtraction angiography has the following advantages:

a) Digital subtraction angiography accomplishes significantly better contrast resolution; it does with a sacrifice of spatial resolution.

b) Firstly, the highly sensitive screening technique for diagnosing for carotids and lower limb vessels.

c) When compared to conventional angiography cost is less.

d) Digital subtraction angiography can be performed routinely on out-patient basis.

e) IV digital subtraction angiography is probably less dangerous than conventional angiographic procedures.

f) Digital subtraction angiography may demonstrate small reconstituted vessels distal to an obstruction not seen on a catheter cut film study.

Disadvantages

a) By comparison to conventional angiography digital subtraction angiography has poor spatial resolution.

b) With digital subtraction angiography biplane capability is harder to achieve.

c) Overlapping vessels have proven to be a significant problem.

d) Patient motion is also problem with digital subtraction angiography.

3. Radionucleotide bone Scintigraphy:

- Bone scanning using Technitium 99m phosphonates is useful in identifying early osteomyelitis.
- Gallium accumulates in areas of active inflammation
- Sequential gallium scan are useful in monitoring the response to treatment for chronic osteomyelitis.

4. Computed tomography

- Well suited for imaging complex articulations and numerous soft tissue structure.
- Can identify and characterize the extent of soft tissue infection.

5. Magnetic resonance imaging

- Detects and display bone marrow alteration in osteomyelitis

- Displays the contrast between soft tissue, medullary tissue and cortex with clarity

Chance of Ischemic rest pain

Ankle pressure	Unlikely	Probable	Likely
Non diabetic	More than 55	35-55	Less than 35
Diabetic	More than 80	55- 80	Less than 55

Prediction of healing of ulcer

Ankle pressure	Likely	Probable	Unlikely
Non-diabetic	More than 65	55-65	Less than 55
Diabetic	More than 90	80-90	Less than 80

Chance below knee amputation healing

Diabetics	Likely	Probable	Unlikely
Calf pressure	More than 65	More than 65	Less than 65
Ankle pressure	More than SO	More than SO	Less than SO

CHART FOR MANAGEMENT OF DIABETIC FOOT

Diabetic foot

↓

Measure SPP, SVR

↓

Healing likely

↙ ↘

Yes No

Arterial surgery not required Angiography

↓ ↓

Rest, antibiotics, Arterial recontrucion

Drainage of abscess ↙ ↘

Yes No

Arterial bypass Primary amputation

MANAGEMENT OF DIABETIC FOOT

MEDICAL MANAGEMENT

CONTROL OF DIABETES

- Patient education
- Dietary modification
- Pharmacological therapy
- Treatment of DKA

Patient education :

Patient education goes a long way in the accurate control of diabetic status.

Dietary modification:

Composition and timing of meals are particularly important for these patients. Caloric goal should be supply roughly about 35 Kcal/kg/day.

Regarding food composition, it is prudent to maintain a balance of carbohydrates, proteins and fat, i.e

Carbohydrates : 60-65%

Protein : 10-20%

Fats : 25-35%

Pharmacologic therapy:

INSULIN REQUIREMENTS AND ADMINISTRATION:

May rise to 1 or 2 units/kg daily acute illness (basal=0.5 or 0.6 units/kg/day). Adjust the insulin dosage in the dextrose insulin infusion based on blood glucose monitoring. An additional single bolus of regular insulin may be given intravenously or subcutaneously for rapid lowering of the blood glucose level to below 300mg/dl.

For total parenteral nutrition, add regular insulin 15-40 units to each litre of solution infused over 8 hour period.

Blood glucose monitoring, four to six time per 24hours, is needed to provide guidance for insulin dosage in all systems of insulin delivery during total parenteral nutrition or insulin resistant stages.

Separate infusions of glucose and insulin are used in a piggy back system (5 to 7.5g of glucose/hr and 1-3 units /hr of insulin by pump) for severe problems or for emergency surgery. Increments of insulin are made when urine is positive: regular blood and urine sugar measurements must be done even after the patient is stabilized.

Split normal dose regimen:

1) Determine fasting blood glucose level

2) Administer insulin, half the usual morning dose is given subcutaneously as NPH

3) Start an infusion of 10 dl 5% dextrose normal saline solution at 1 dl/hr.

4) During the operation, a separate intravenous line for IV fluids may be needed.

5) Monitor blood glucose levels intra operatively during long operations, in recovery room, and four times daily thereafter.

6) If the blood glucose level greater than 250mg/dl give 10% to 15% of the total daily insulin requirements as regular insulin subcutaneously.

7) Give a second dose of NPH half the usual dose, 8-12 hours later.

8) Continue administrating IV dextrose 5% solution with 20 meq of potassium chloride. Total daily dextrose intake is 150 g or more. Potassium chloride additions are based on potassium monitoring.

9) A split dose of NPH should be continued daily after surgery. Add regular insulin if the blood glucose level is elevated.

10) For unstable patients, change to the dextrose insulin infusions method or to separate iv insulin infusion.

11) Resume the usual insulin regimen when oral feeding has started.

GLUCOSE INSULIN INFUSION METHOD FOR INSULIN DEPENDENT DIABETICS:

Schedule surgery in the morning, if possible obtain plasma and electrolyte determinations. Start an infusion of 10 dl 5% dextrose in 0.9% sodium chloride (DNS) at 1 dl /hr with regular insulin added as follows.

Daily insulin requirement units/24hr	Insulin (units/1 D5 NS)	Infusion rate units/hr
40 or less	10	1.0
40 – 80	15	1.5
Over 80	20	2.0

The infusion rate may be increased during the operation if the fluid requirements are elevated.

Check the blood glucose level in the recovery room and four times daily thereafter. Adjust the insulin dose based on the blood glucose levels follows:

Blood glucose levels	Adjust insulin dose
< 90 mg/dl	Reduce dose by 5 units
90- 199	Continue same dose
200- 280	Add 5 units to infusion
>280	Add 10 units to infusion and give 5 units subcutaneously or as IV bolus

Add potassium chloride , 20 meq to infusion post operatively .If serum potassium level <3.5, increase potassium chloride to 40 – 80 meq/L; if serum potassium level >5, no potassium chloride is added.

Post operative management: Administer dextrose insulin potassium infusion at rate to maintain hydration usually 3L daily in half normal saline solution; Add multivitamin preparation. Adjust insulin and potassium chloride based on daily monitoring data.

Glucose – insulin infusion for non- insulin dependent diabetics:

Monitor the plasma glucose level in recovery room and four times daily thereafter.

Blood glucose level	Adjust insulin dose
< 90 mg/dl	No insulin
90 – 199	Continue same dose
200- 280	Add 5 units to infusion
>280	10 units infusion and give 5 units subcutaneous

Add potassium chloride (20 meq) to the dextrose – insulin infusion post operatively.

Continue the dextrose – insulin – potassium infusion until oral feeding is resumed.

Adjust regular insulin and potassium chloride additions based on monitoring data.

At discharge from hospital, when we contemplate on reinstituting dietary therapy and or oral hypoglycemic drugs for diabetic control.

TREATMENT OF DIABETIC KETOACIDOSIS:

This should include-

- Restoration if volume with appropriate fluids and electrolytes as fluid deficit of 4-5 litres.
- Reversal of acidosis and ketosis.
- Control of plasma glucose
- Stabilization of patients in shock and management of coma should proceed without delay.

1) INSULIN REPLACEMENT

Initially started with a loading dose of 0.3units /kg as a bolus to prime tissue insulin receptors followed by 0.1 unit/kg/hr; either continuously infused or given intramuscularly doesn't improve.

2) CONTROL OF INFECTION:

In most instances, the initial choice of antimicrobial agents is made prior to knowledge of microbiologic culture results. The decision will be based on the most likely probability; the severity of the illness may also play a

role in the choice of antimicrobial therapy. The antibiotic can be changed based on culture reports.

Mild infections: (minimal cellulitis, mild purulence in a pre-existent ulcer, or an infected blister)

If there are no clinical manifestations of sepsis, monoantibiotic therapy may be instituted while awaiting culture and sensitivity reports. In the absence of necrotic tissue, foul smelling discharge and frank gangrene, it is more common to isolate single microorganisms and anaerobes are relatively uncommon (24). In this gram + aerobic cocci are usually dominant organisms.

These include - Staphylococcus aureus, Coagulase negative staphylococci, Nongroup D streptococci, Enterococci..

GENERAL SUPPORTIVE CARE:

a) To increase local tissue perfusion by administrating tab Pentoxyphylline 400mg/PO/TID for 8-10weeks.
b) General improvement in nutrition by replacing vitamins orally or parenterally along with a balanced caloric diet.
c) Correction of anemia by blood transfusion (preferable instead of oral iron)

SURGICAL MANAGEMENT:

1. TREATMENT OF CORNS AND CALLUSES

Corns calluses are due to friction and pressure, most often from improperly fitting shoes and stockings. Wear proper fitting shoes. Do not tear it off. Do not cut corns or calluses unless there is underlying infection.

Excision and drooling can be done and granulation scraped if there is infection.

2. TREATMENT OF FISSURES:

Fissures are more frequently caused by dry skin especially in the area of the heel around calluses or in the presence of chronic fungal infections. They must be treated with regular applications of moisturizing and/or antifungal creams like Eucerin, hydrated lanoline, antifungal like Tolnaftate.

3. TREATMENT OF BLISTERS:

Blisters must be opened or unroofed to relieve the pressure on the underlying tissue and to permit culture of the exudates.

4 . LOCAL WOUND CARE:

This is directed at protecting the wound from further trauma and contamination, to facilitate wound drainage and to provide antibacterial

coverage that also serves to clean and debride the ulcer. The primary goal in the treatment of diabetic foot is to obtain wound closure.

MINOR ULCERS

For a minor ulcer topical medication covered with a dry gauge pad changed once or twice daily is adequate. Topical medications frequently used are 5 % Povidine iodine solution, 1 % silver Sulphadiazine cream, Gentamicin cream, Bacitracin ointment, Hydrogen peroxide, EUSOL.

MAJOR ULCERS

Infected lesions need hospitalization and adequate debridement.

METHODS:

- Superficial debridement
- Incision and drainage of abscess
- Debridement of deep infection and necrosis

Debridement

These Principles to be followed during a debridement.

i. All areas of dead tissue should be removed.
ii. The margins of the ulcer should be trimmed up to the point, where it bleeds freely.
iii. An open wound particularly near a joint should be probed to see for communication.
iv. Radical debridement including the dorsal skin should be performed if skin of the dorsal surface following cellulitis get necrosed.
v. Palmar surface drainage with amputation of the necrotic toes in case of central palmar space abscess.
vi. Tissues left after debridement should not be damaged by forceps or any other crushing instruments.
vii. If pus or fluid is present it should be sent for culture and sensitivity.

TREATMENT OF NEUROPATHIC ULCERS:

Neuropathic ulcer involving Heel

The main objective in the treatment of neuropathic ulcers is to reduce the mechanical stress of the insensitive feet is by giving rest, maintaining ambulation, elevation of foot and relief of pressure.

The cast is applied in such a way that it spread the excess pressure throughout foot and lower leg and allowing the ulcer to heal quickly. It also protects the foot from further damage, reduce edema. It should be reapplied weekly.

The main disadvantages of using a total contact cast are joint stiffness and muscle atrophy on long term use. An improperly applied cast lead to abrasions and skin ulcerations. They cannot be used in presence of active infection, foot swelling, obesity and ABI <0.45.

Granulation Tissue

SOFT TISSUE TECHNIQUES TO SALVAGE DIABETIC FOOT

1. Skin grafts are 2 types

a) Split skin graft- epidermis & portion of dermis only

b) Full thickness graft - epidermis and all of dermis

Split Skin Grafting

2. Local flaps- 2 types

a) Rotation flap- rotated around a pivot joint

- Transposition flap
- Limberg flap
- Z plasty
- Interpolation flap
- Island flap

b) Advancement flap – advanced forward from their base

- V.Y f

3. Muscle/musculocutaneous flap

- ADM flap

- Abductor hallucis brevis flap
- FOB flap

4. Faciocutaneous flap

- Lateral Calcaneal artery FC flap
- Plantar flap

RECONSTRUCTIVE ARTERIAL SURGERY:

It should be considered only after measuring the ankle systolic pressure by Doppler flow meter. If an ankle pressure is < 2/3 of the arm pressure, there will be large vessel disease where reconstructive arterial surgery for limb salvage is worth trying.

The surgery carried out will be depending on the extent of arterial occlusion which can be find out by arteriogram.

INDICATIONS OF PERIPHERAL VASCULAR SURGERY IN THE DIABETIC FOOT- Nocturnal pain, Rest pain, Foot ulcers not responding to treatment, Infections not responding to treatment, Incipient gangrene, Severe disabling intermittent claudication.

In a patient with arterial insufficiency arterial surgery should be considered in the following- Claudication which significantly interferes with work, Rest pain and an area of gangrene which doesn't heal or can t be removed surgically, treated with success by a local foot operation.

NON-OPERATIVE CORRECTION OF SELECTED ARTERIAL BLOCKAGES: ANGIOGRAPHY

Transluminal balloon dilatation is done by means of a small balloon catheter which is carefully introduced into site of stenosis and distended under fluoroscopy, thereby relieving the obstruction.

MINOR AMPUTATIONS:

The principles regarding local amputations are:

1. Infected areas should be widely opened
2. Tourniquet should be avoided.
3. No place for small incisions, as if the infections usually will be deep which requires wide incisions and excision of devitalized tissues.
4. The other foot must be taken care of because this may be the only one the patient will have.

VARIOUS TYPES OF LOCAL AMPUTATION

1. Transphalangeal amputation of a toe
2. Amputation of a single toe and Ray amputation
3. Transmetatarsal amputation
4. Syme's amputation

TRANSPHALANGEAL AMPUTATION OF A TOE:

This is the most commonly performed amputation. If there is evidence of arterial insufficiency, adequate collateral circulation as shown by a venous filling time of 20 sec or less is necessary. The lesion must be in the distal 1/3 of the toe, leaving reasonably healthy skin at the time of incision.There should be no dependent redness of the proximal part of the toe and all cellulitis and lymphangitis should have cleared prior to operation.

RAY AMPUTATION

This operation removes one toe and distal half metatarsal shaft including the head through a racquet shaped incision, this leaves a residual space which cannot be surgically closed and healed by secondary intention with scar formation which eventually pulls the adjacent metatarsal together. The adjacent joint capsules are avascular structures which further delay healing, the operation is therefore reserved for those patients with quite good collateral circulation. This procedure is more useful in the neuropathic rather than the ischemic foot and hence mainly done for ulcers under the first or fifth metatarsal heads or beneath one of the other metatarsal head

TRANS METATARSAL AMPUTATION

Transmetatarsal amputation may have to be done if more than one toe is involved or there is persistent recurrent plantar ulcer. In this, one removes all of the toes and metatarsal heads, using a plantar flap resembling a Turkish slipper for closure.

Infection must be controlled and the skin of the dorsum and the sole of the foot must be healthy. It may take upto 3 weeks of care in the hospital to prepare the patient for the operation. Pulses need not to be present in the foot but the collateral with a venous filling time of less than 25 seconds and absence of dependent redness at the level of incision.

SYME 'S AMPUTATION

This is indicated where more than one toe is involved, sepsis is confined to distal half of the foot and the large vessel disease is absent. The role of this amputation is diabetic foot however still remains controversial. The main reason for its advocacy is that the patient will be able to walk on his own leg (feeling of earth) and without shortening of his limb.

BELOW KNEE AMPUTATION:

We select the below knee level if the patient is expected to use a prosthesis, if the area of gangrene is below the ankle with a demarcation

at or below the ankle level. The amputation is usually accomplished slightly below the mid lower leg level using short and equal anterior or posterior flaps, dividing the fibula one inch higher that the tibia, and the tibia at a suitable level to permit easy closure

ABOVE KNEE AMPUTATION

The operations most commonly performed are:

1. Through knee disarticulation
2. Supracondylar articulation
3. Mid – thigh amputation

The first two operations are likely to heal well due to presence of collateral vessels in the area of the knee joint and the subcutaneous tissue. Both operations involve division of only small amount of muscle tissues which has the practical advantage of reduced anaerobic sepsis. Both have a disadvantage of difficulty in fitting a proper prosthesis. Of the three the supracondylar amputation offers the ideal stump.

RECENT TRENDS

Vacuum assisted closure

Clean, non- healing deep cavity wounds may respond to repeated treatments by application of negative pressure under an occlusive wound dressing (VAC).

Vaccum assisted closure

Hydrotherapy

Intractable, infected, cavity wounds so metimes improve with hydrotherapy using saline pulse lavage under pressure.

Skin grafts

The autologous skin graft is the standard criterion for viable coverage of the partial thickness wound. The graft could be harvested under LA as an outpatient procedure. Meshing the graft allows wider coverage and promotes drainage of serum and blood.

Tissue cultured skin substitutes

Dermagraft: is a cryopreserved human fibroblast- derived dermal substitute produced by seeding neonatal foreskin fibroblasts onto a bioabsorbable polyglactin mesh scaffold. It is used for managing full thickness chronic diabetic foot ulcers, which is not appropriate for infected ulcers, those involving bone or tendon or those having sinus tracts.

Apligraf (Organogenesis): is a living, bilayered human skin substitute. It is not used for infected ulcers, with involvement of tendon or bone or those having sinus tracts.

Surgical wound closure

Delayed primary closure of a chronic wound requires well-vascularized clean tissues and tension free apposition. It usually requires undermining and mobilization of adjacent tissue planes by creation of skin flaps or myocutaneous flaps.

Hyperbaric oxygen treatment:

This is rarely used, not a substitute for revascularization. In the presence of intractable wound and associated non correctible ischemic arterial disease, the hyperbaric oxygen therapy may be beneficial

REHABILITATION

The following are done in our study:

a) Protective shoes – protect from further trauma and helps in effective weight bearing.
b) Crutches – after amputation, gait training is given with this
c) Prosthesis – Absolute minimum prosthetic requirement for the AKA is a wheel chair or a set of walking crutches.

FOLLOW-UP

Health education regarding the foot care and diabetic control were given at the time of discharge. All the patients were asked to review after 1 week during the follow up, diabetic status was checked and detailed general and local examination was done.

PREVENTION (DIABETIC FOOT CARE)

Care of foot takes place at three level:

1. The patient must take routine measures to take care of his foot
2. Early lesions require expert care either from a podiatrist or from an experienced doctor in the care of the patient.
3. Advanced lesions require specialized care.

The best way is to make use of multidisciplinary professionals who are committed to limb salvage. Team members involved are- Physician,

Nurse, Endocrinologist, Podiatrist, Neurologist, Vascular surgeon, Orthopedist, Physiotherapist, Social workers, Home care nurse.

Patient education is important in case of diabetic foot.

The advice given to patient include (25)

- Do not smoke
- Inspect the feet daily for blisters, cuts and scratches. With the use of a mirror can see the bottom of feet, also check in between the toes.
- Wash feet daily, dry carefully, especially between the toes.
- Avoid extreme temperature. Test water with hand, elbow or thermometer before bathing
- If feet feel cold at night wear socks. Do not apply hot bottle water or heating pads.
- Do not walk on hot surfaces.
- Do not walk barefoot or soak feet.
- Do not use chemical agents for removal of corns, calluses
- Inspect the feet daily for foreign objects, nail points, rough areas
- Do not wear sandals with thongs between the toes
- In winter season, wear wool socks and protective gear such as fleece lined boots.

MATERIALS AND METHODS

TITLE OF THE STUDY: A STUDY OF PATHOPHYSIOLOGY, MANAGEMENT AND FACTORS INFLUENCING DIABETIC FOOT ULCERS AMONG DIABETIC PATIENTS.

STUDY DESIGN : Observational study

METHODOLOGY

Sample size –

$n = (Z\alpha)^2 pq / d^2$

$Z\alpha^2 = 1.96$

Where p- prevalence and q = 100 – p;

d = maximum allowable limit of error over prevalence; i.e. (20% of p)

p = 29 % ; q =71% and d = 20% of p = 5.8

Hence sample size n = 250

A observational study was conducted on 250 diabetic foot ulcer patients attending surgical op/ emergency department (1 B) in CMCH over a period of 1 year, from July 2016- July 2017.

STUDY POPULATION

INCLUSION CRITERIA

250 patients with age > 13 years presenting to surgical op/ emergency department (1 B) with diabetic foot ulcer

EXCLUSION CRITERIA

- Patients below 13 years
- Pregnant females
- Psychiatric patients.
- Diabetic patients who have ulcer other than diabetic ulcer for example traumatic ulcer, venous ulcer.

METHODS OF COLLECTION OF DATA

After registration and admission , detailed clinical history of patient taken, followed by a detailed clinical examination.

The name, age, sex, address, profession of the patient is noted. The symptoms and clinical features are recorded in chronological order.

Clinical history includes the following points- Known case of diabetic or not, Duration of diabetes, regarding the treatment received if any, Family history of diabetes, any history of injury, Local symptoms such as

swelling, pain, wound, discoloration andPersonal habits such as smoking and alcoholism.

General examination of the patient includes all vitals and other system examination.

Clinical features of neuropathic foot are- Warm with intact pulses, Diminished sensations, callus, Ulceration, Sepsis, Local necrosis, Edema, Charcot's joints.

Clinical features of ischaemic or neuro-ishaemic foot are Cold with absent pulse, diminished sensations, Ulceration and Necrosis or gangrene

In the examination of the feet, the following points are to be noted- Types of lesion and extent, evidence of any predisposing factors, any changes suggestive of neuropathy or vascular involvement

The neurological status of the lower limb assessed to rule out diabetic neuropathy. All the sensations, power, reflexes, and neurological deficit were noted.

Vasculopathy of the limb was found by assessing Colour of limb: normal, pale, purpule, black, local temperature: normal or cold and the pulsations of the lower limb: dorsalis pedis, posterior tibial, popliteal and femoral artery.

OUTCOME

- Enhancing the importance of patient's knowledge in self-care practice and regular diabetic foot evaluation.
- Reduce the morbidity of patients with diabetic foot ulcer and enhance early healing of ulcer.
- Early rehabilitation of the patients with diabetic foot ulcer.

OBSERVATIONS AND RESULTS

An analysis of 250 cases of diabetic foot was done. These cases were treated in different surgical units in the Department of Surgery, Coimbatore Medical College Hospital from July 2016 to June 2017.

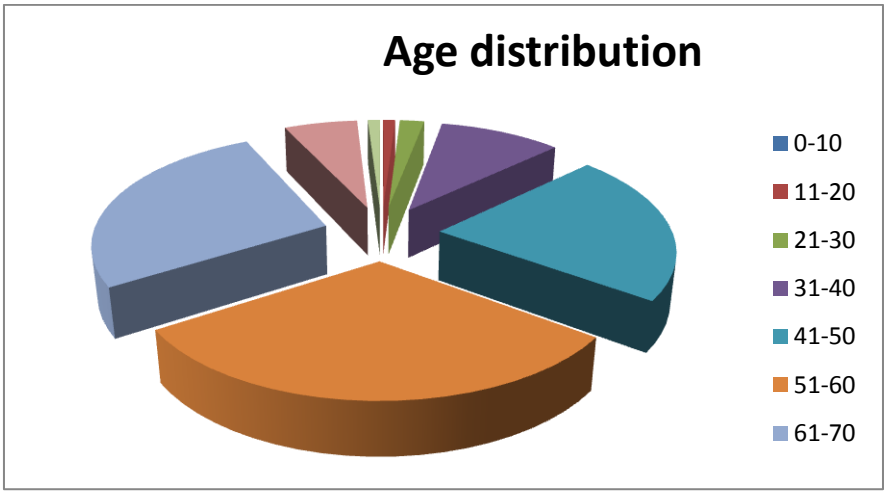
In our study 250 patients of diabetic foot lesions were studied. In 35 patients only incision and drainage and fasciotomy was done, healing of wound occurred without complications. In 125 patients debridement was done as the definite treatment, as a groundwork to amputation. Skin grafting was well thought-out in 40 patients once the wound was clean and granulating.

In our study amputation rate was 20%. Out of these, patients underwent Above Knee or Below Knee Amputations or minor amputations. In 40 patients major amputations like Below Knee and Above Knee amputations done. 15 Patients had wound infection and suturing gaping. For these patients with wound gaping secondary suturing was done. In 10 patients, Above Knee amputations was done, 5 patients stump was closed primarily and in 5 patients Gullettine amputation was carried out. A total of 10 patients died because of various complications of diabetes during the line of treatment.

1.Age distribution

Table :-1 Showing the Age distribution

Age (years)	No. of patients	Percentage (%)
0-10	-	-
11-20	2	1
21-30	5	2
31-40	25	10
41-50	55	22
51-60	80	32
61-70	66	26
71-80	15	6
81-90	2	1
Total	250	100

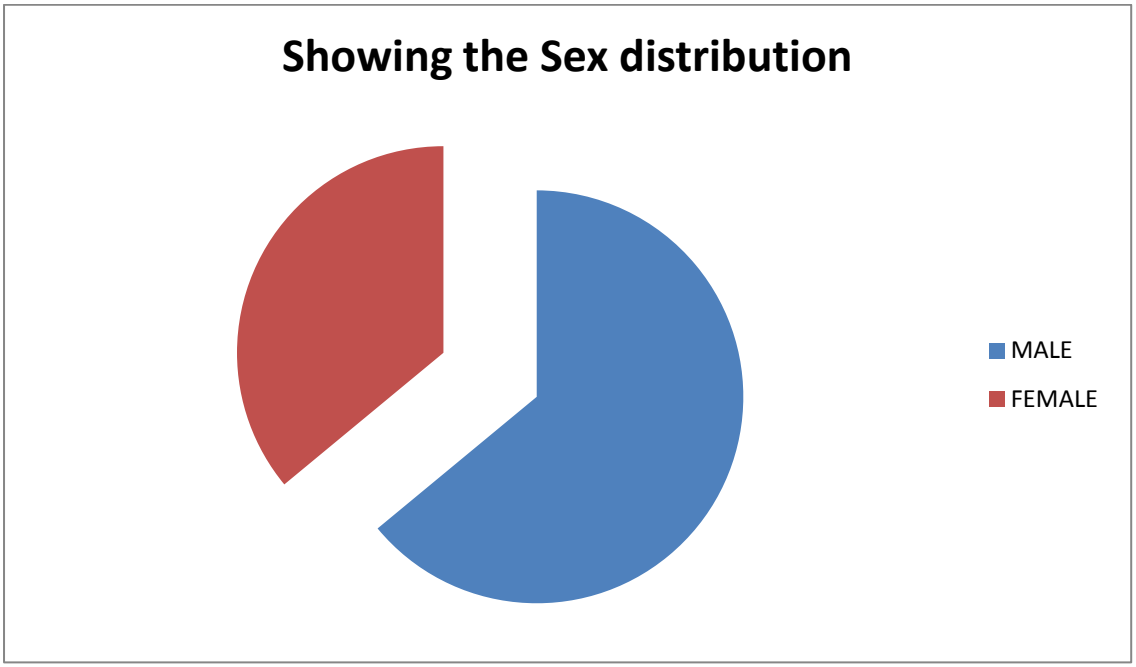


Of 250 cases studied, youngest patient was 19 years and oldest was 84 years of age. Highest number of cases was found in the age group 51-60 years (31%) followed by 61- 70 years (27%). Maximum number of diabetic foot i.e 80% are between the age group of 41-70 years.

2. Sex

Table:- 2 Showing the sex distribution

Sex	No. of cases studied	Percentage(%)
Male	160	64
Female	90	36

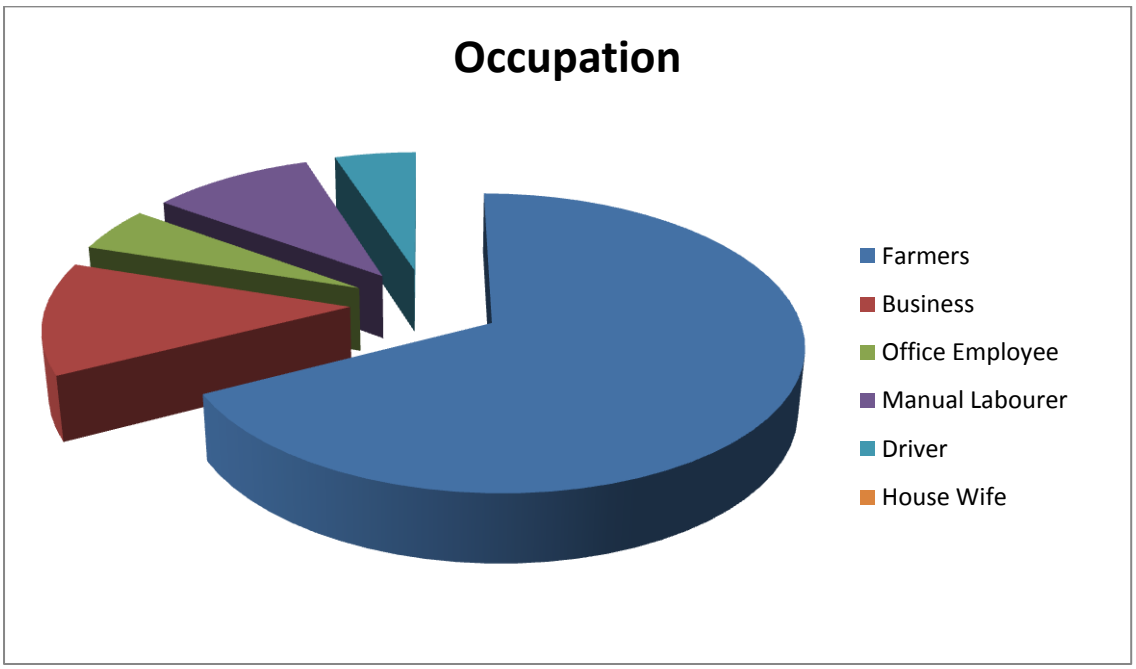


Of the 250 cases studied in this series, 160 (64%) cases were male and 90(36%)

3.Occupation

Table 3:- Occupation

Sl.No.	Profession of patient	No. of cases	Percentage
1	Farmers	135	54
2	Business	25	10
3	Office Employee	10	4
4	Manual Labourer	20	8
5	Drivers	10	4
6	Housewife	50	20
	Total	250	100

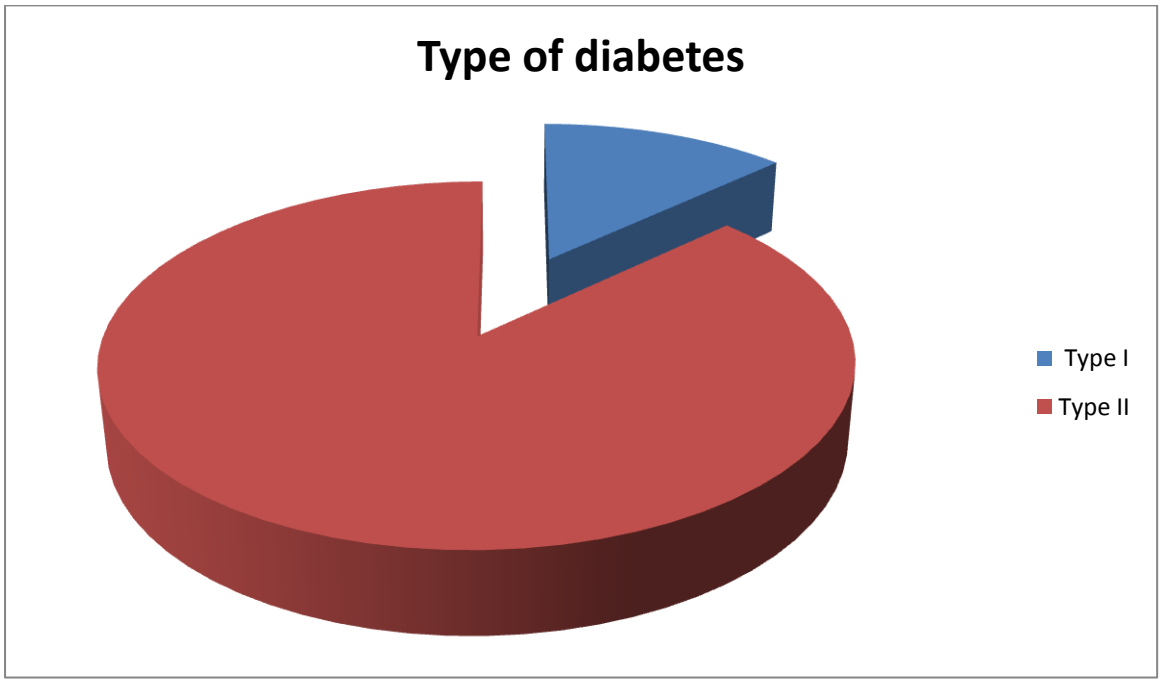


In this study maximum patients were farmers (54%) and minimum patients were drivers and office employee (4%).

4.Type of diabetes

Table:- 4- Type of diabetes

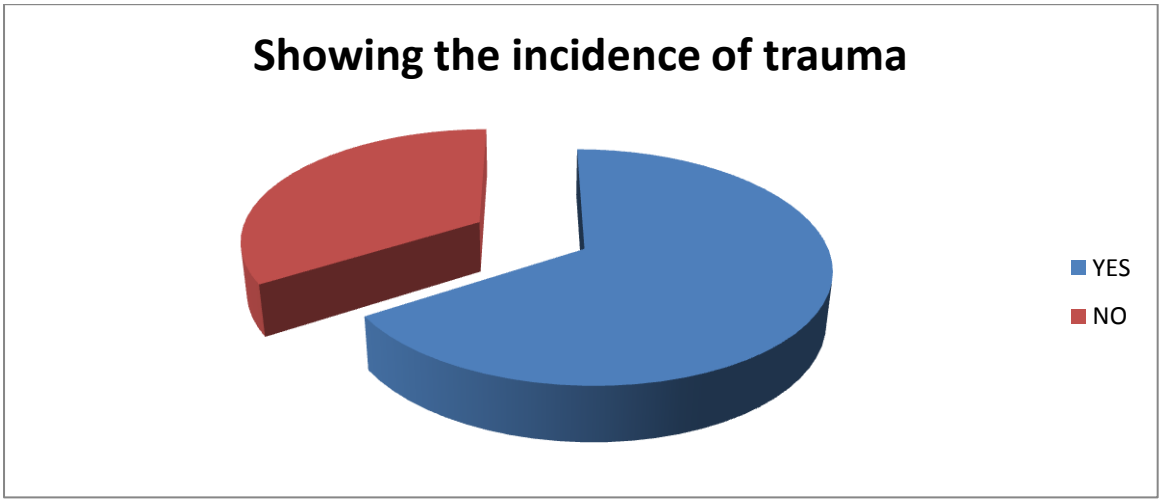
Type of diabetes	No. of patients	%
I	35	14
II	215	86



In this study 35 patients had type I diabetes, remaining 215 patients had type II diabetes.

5.Incidence of Trauma

Chart 5:- Incidence of Trauma

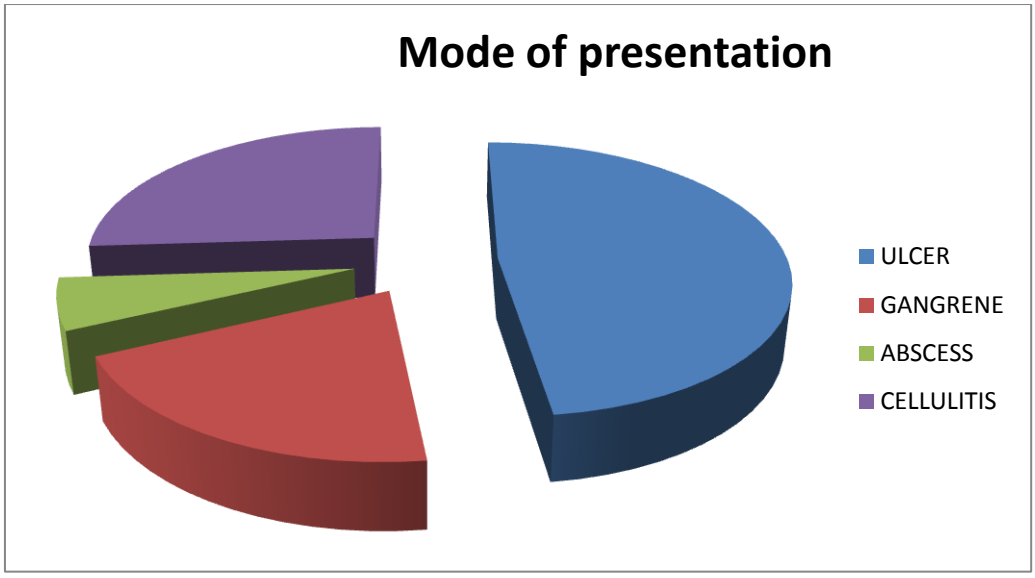


In this study 64 cases exposed a history of some kind of trauma before the onset of lesion.

6. Mode of presentation

Table 6 :- Showing incidence of various types of lesions.

Mode of presentation	No. of cases	%
Ulcer	120	48
Gangrene	50	20
Abscess	15	6
Cellulitis	65	26



The different types of lesions seen including ulcer, cellulitis, abscess and gangrene. Most of the patients present with more than one lesions. Only major lesion is considered here. Ulcer was the major lesion seen and is present in 120 patients. 15 patients presents as a abscess was the least common lesion.

In above patients x-ray of 30 cases showed changes of Osteomyelitis. 15 patients present with Charcot's joint.

Doppler studied in 10 patient showed atherosclerotic changes with low volume flow in anterior and posterior tibial arteries.

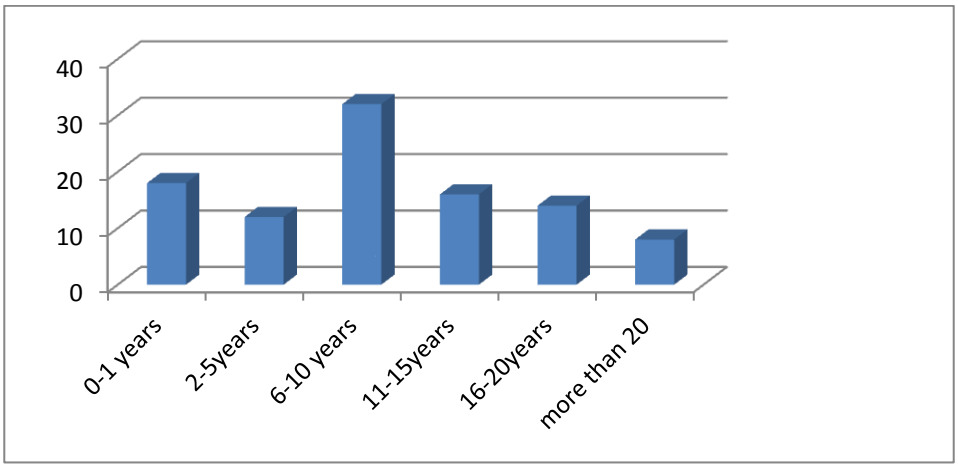
7. Duration of diabetes

Duration is not accurately known, as few patients were unaware of being diabetics and were newly diagnosed as suffering from diabetes on admission with complaints of non- healing ulcer or other complications of Diabetes.

Table 7 :- Showing duration of Diabetes

Duration of diabetes in years	No. of patients	%
0-1	45	18
2-5	30	12
6-10	80	32
11-15	40	16
16-20	35	14
>20	20	8
Total	**250**	**100**

Showing duration of diabetes:-



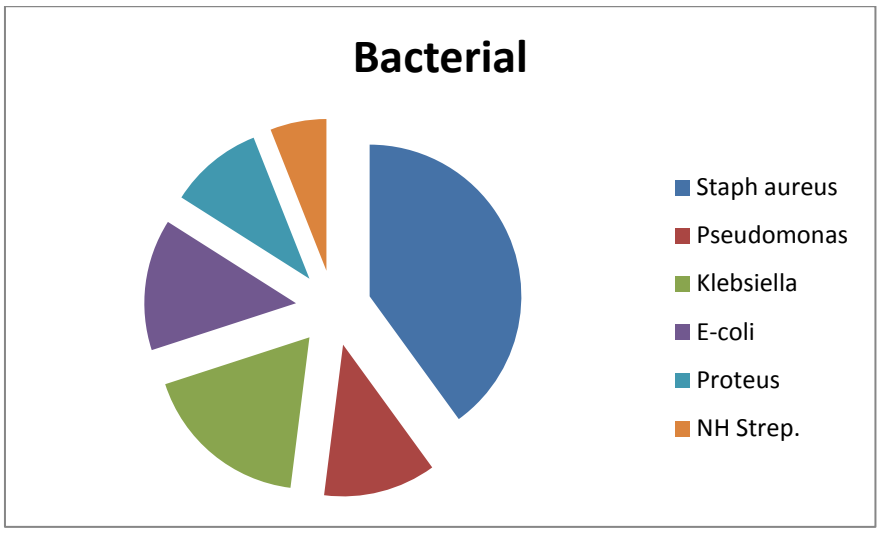
In our study 18% presented with duration less than or equal to 1 year. Most of these patients were diagnosed post admission. Only 20 patients had diabetes of more than 20 years. Maximum 80 patients in our study were diabetes of 6- 10 years.

In the present series 10 patients were detected as a diabetic at the time of admission.

8.Culture and Sensitivity

Table 8 :- Culture and Sensitivity

%	Bacterial	No. of cases
40	Staphylococcal aureus	100
12	Pseudomonas	30
18	Klebsiella	45
14	E- coli	35
10	Proteus	25
6	Non-hemolytic streptococci	15

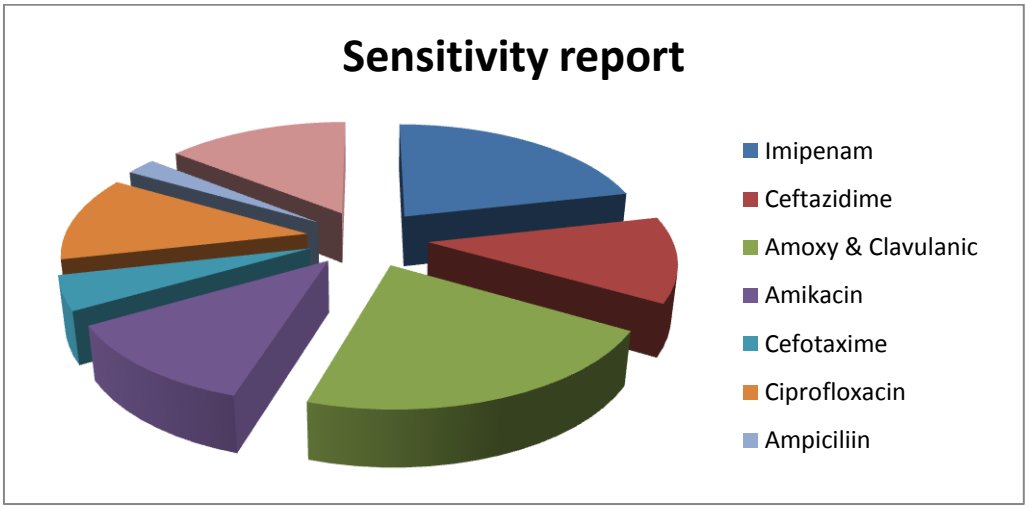


In our study majority of septic lesions yielded Staphylococcus aureus in about 40% on culture of pus. Other organisms were isolated are Pseudomonas 12%, Klebsiella 18%, E-coli 14%, Proteus 10%.

9. Sensitivity report

Table 9:- Showing Antibiotic Sensitivity according to Culture.

Antibiotics	% of patients
Imipenam	45
Ceftazidime	25
Amoxycillin and Clavulanic acid	45
Amikacin	25
Cefotaxime	10
Ciprofloxacin	25
Ampicillin	5
Vancomycin	30

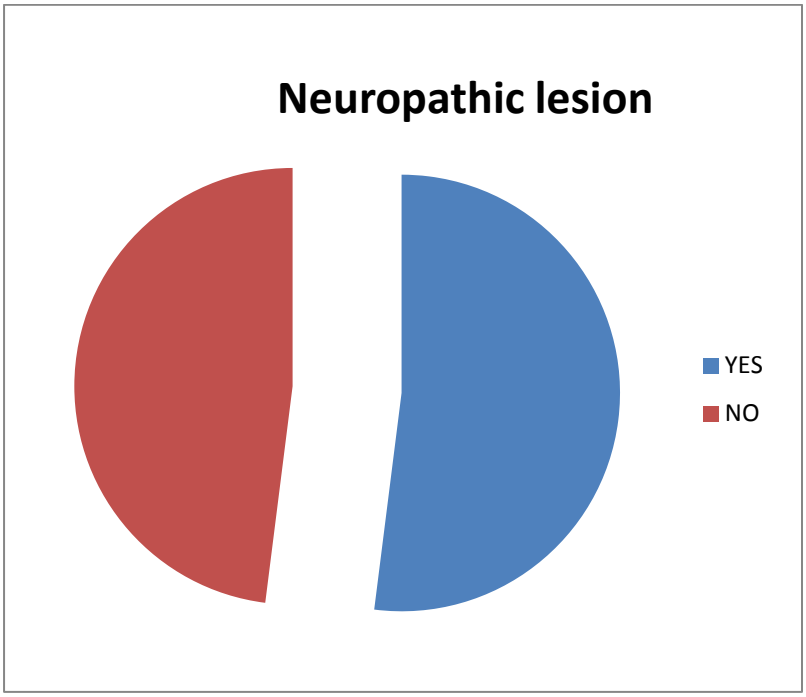


Imipenem and Amoxicillin & Clavulanic acid were sensitive against most of the organisms as they cover a wide range of organisms.

10. Neuropathic lesions

Table 10:- Neuropathic lesions

Neuropathic lesions	No. of cases	%
Yes	130	52
No	120	48

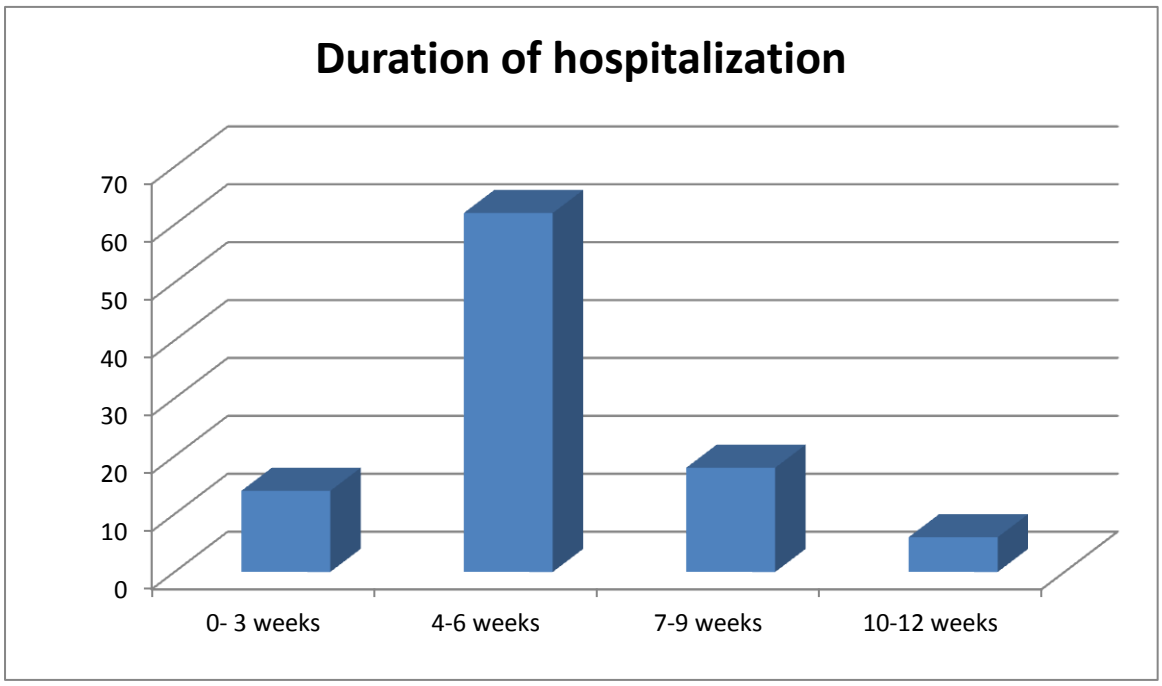


In the present study 130 cases were found to have neuropathy. Patients with neuropathy varied from 35-80 years. Majority had history of diabetes more than 5 years. This shows that peripheral neuropathy is common in long standing diabetic patients. 50 patients had gangrene.

11. Duration of hospitalization

Table :-11- Duration of hospitalization

Stay in hospital	No. of patients
0-3 weeks	35
4-6 weeks	155
7-9 weeks	45
10-12 weeks	15

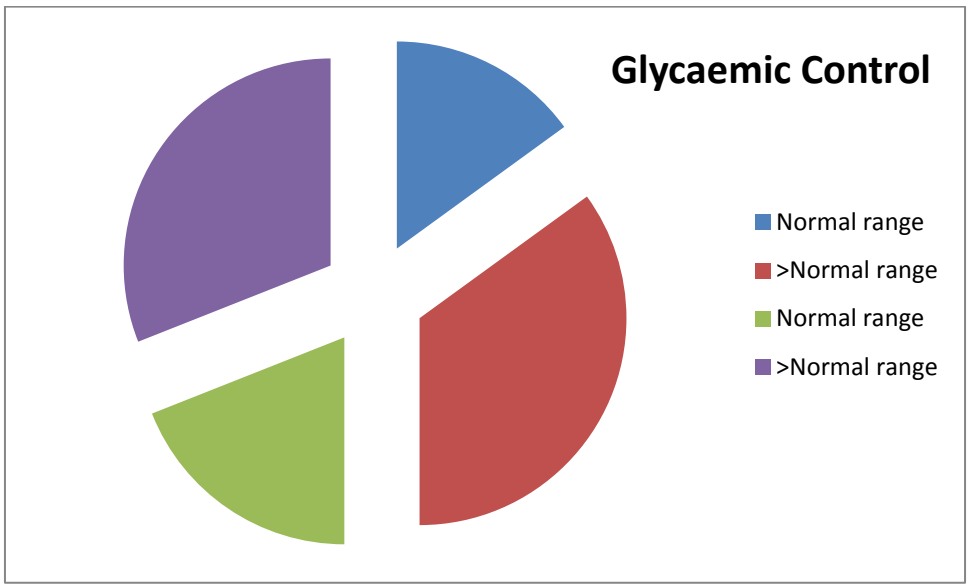


In this study minimum stay in hospital was 1 week (7 days) and maximum was 12 weeks (84 days). Most of patients stayed in hospital form 4-6 weeks.

12. Investigations

Table 12:- Showing blood sugar level at the time of admission

Blood sugar			
Random blood sugar		**Fasting blood sugar**	
Normal range	>normal range	Normal range	>normal range
75	175	95	155

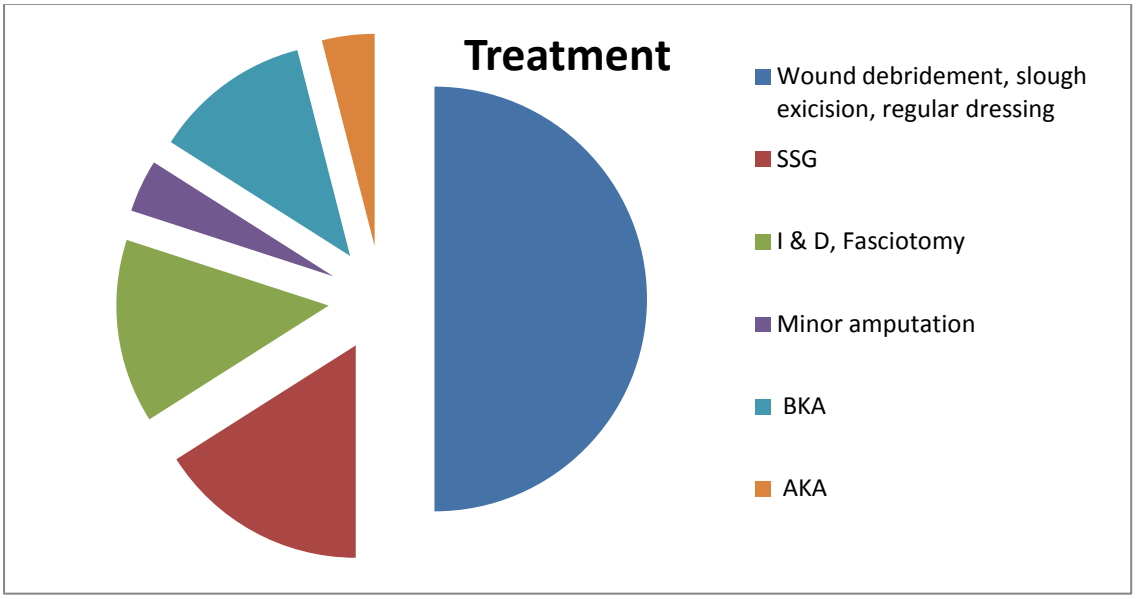


In this study at the time of admission 175 patients had RBS more than normal and 75 patients had RBS within normal range. While FBS at the time of admission in the same age group more than normal in 155 patients and within normal range in 95 patients.

13.Treatment

Table 13:- Treatment

Treatment	No. of cases	%
Wound debridement, slough exicision, regular dressing	125	50
SSG	40	16
I & D, Fasciotomy	35	14
Minor amputation	10	4
BKA	30	12
AKA	10	4

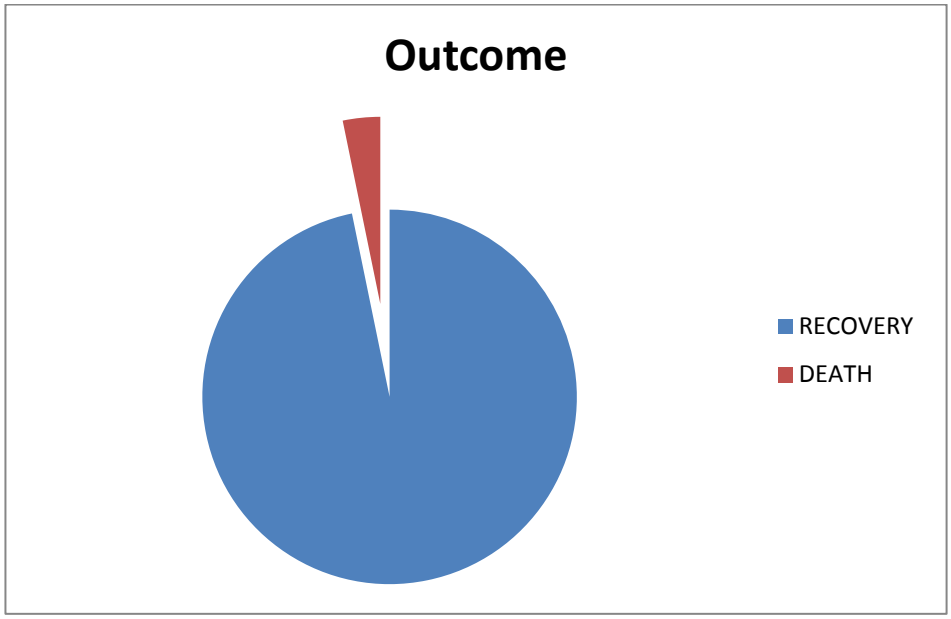


In this series 125 cases were managed by daily dressing and wound debridement, and slough excision. 40 patients were treated with SSG, 35 patients under went Incision & Drainage for abscess and some of them fasciotomy. Minor amputation was done in 10 cases. BKA was done in 30 cases and AKA in 10 cases. In most of the cases, limb was salvaged by conservative treatment and minor amputation.

14. Outcome

Table 14:- Outcome

Result	No. of cases	%
Recovery	240	96
Death	10	4

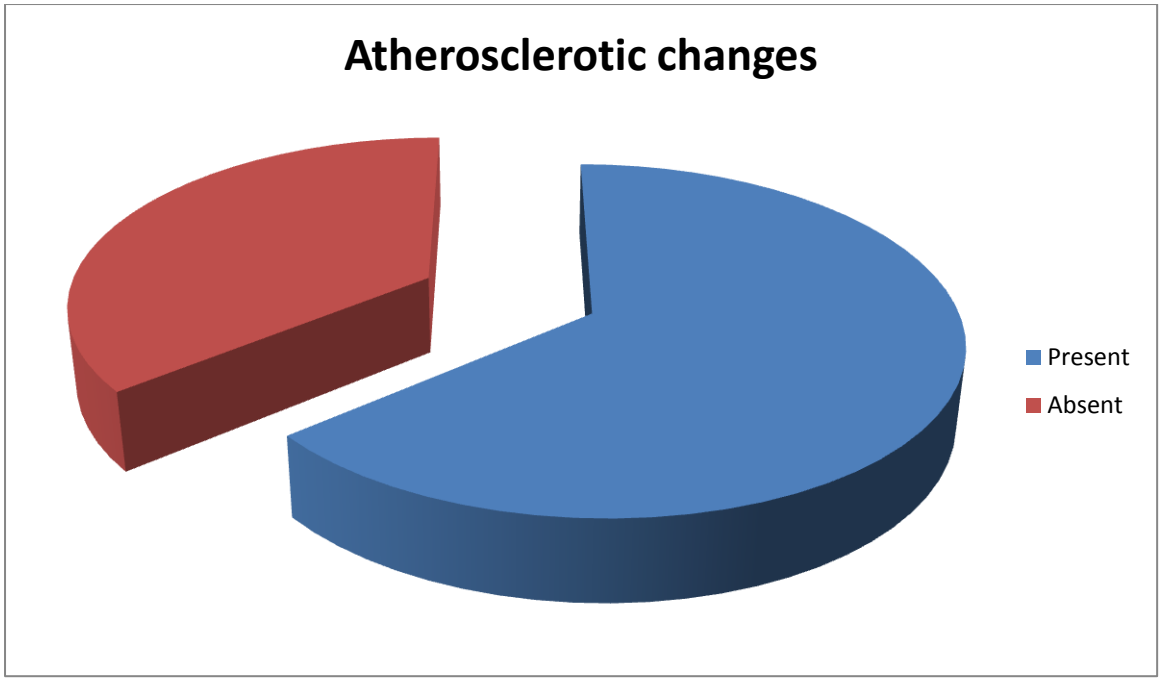


In the present study 240 patients recovered from their lesion after treatment while remaining 10 patients died due to various complications.

15. Atherosclerotic changes.

Table 15:- Atherosclerotic changes.

Result	**No. of cases**	**%**
Atherosclerotic changes present	160	64
No atherosclerotic changes	90	36



Out of 250 patients with diabetic ulcers 160 patients showed atherosclerotic changes in major arteries of lower limb which was proven categorically by an arterial Doppler color examination.

DISCUSSION

Diabetes is a common problem. It affects about 16 million people of all ages and is a major cause of end stage renal disease, blindness and peripheral neuropathies. The disease is chronic and affects the metabolism of carbohydrate, protein, fat, water and electrolytes.

Many diabetic ulcers are ignored because they may produce few symptoms and their importance is not valued by patients and secondly it falls between specialities. Not being entirely in the surgeon or physician domain. Reduction in the occurrence and prompt treatment of foot ulceration would ultimately lead to drop in the amputation rate in diabetic patients.

Without doubt, the problem of foot infection in diabetic patient population is costly to both the patient and society. As a result, health care expenditure and medical hard work must be directed education and prevention, early detection and prompt therapy of food infection.

Total 250 cases of diabetic foot were included in this study. Following is the result of my study conducted at our hospital from (duration of study)

The analysis of this study is as follows.

AGE-

Prevalence of diabetes is greater in persons over the age of 50 years. Pedal infection is a devastating and severe complication of diabetes seen often is elderly patients.

In our study of 250 patients age was ranging from 19 years to 84 years. It was found that age group 51-60 years, had the highest number of 80 patients.

SEX

Most of the diseases have the male predominance, this is same with diabetic foot also. In our series 64% patients were males and 36% patients were females.

The following may be the reason for the male predominance are exposed to the external environment more than females hence they are likely candidates for injury leading to ulcer formation. Alcoholism and smoking habits are more common in males and this may be an important factor. Barefoot walking is more common in India this also contributes to the foot ulceration.

OCCUPATION

As seen in our study, farmers had more incidence of diabetic foot lesions because of unawareness of having Diabetes , lack of foot care and

poor management of diabetes. It is evident that the surgical complications of patients whose profession exposes them to the risk of trauma and injuries making the vulnerable to the complications of diabetic foot.

TYPE OF DIABETES

As per literature foot ulcers have been more in the patient with NIDDM (86%) as per our study. NIDDM age group is being elder and foot complications are more in these patients.

History of trauma

64 cases in this series had history of trauma, before the onset of foot lesion.

In majority of the cases of surgical complications of diabetes, some kind of trauma is the beginning of the problem. This is because of three factors, they are ischemia:- due to ischemia the parts, which are traumatized will be underperfused, thus impairing the healing process.

Neuropathy :- Result in loss of sensation and the patient will be unaware of the injury and neglects it.

Hyperglycemia :- acts as a perfect medium for the multiplying organisms.

Neuropathy which is seen more than 50% of diabetes of long standing duration is considered at to be the single most major course of ulceration.

In present study neuropathy changes seen in 52 cases, ischemic complications was noted in 20 cases and infective complication of foot noted in all cases.

TYPE OF LESION:-

In our study of foot lesions are Cellulitis, Abscess, Ulcer and Gangrene. In some patients there was more than one lesion. Ulcer (48%), Cellulitis (26%), Gangrene (20%), Abscess (6%). Ulcer was the commonest presentation.

DIABETIC STATUS:-

In our study 96% patients were known diabetics. Other 10 patients were diagnosed after hospital admission to hospital.

DURATION OF DIABETES:-

In our study mean duration of diabetes mellitus is 12 years.

CULTURE & SENSITIVITY:-

In our study pus was sent for culture and sensitivity in 120 patients. In most of patient's more than one organism were grown on culture.

In our study staphylococcus aureus (40%), E Coli (14%), Klebsiella (18%), Proteus (10%), Pseudomonas species (12%) were commonly isolated organisms.

Imipenem, Meropenem and Cefepime were the most effective agents against gram negative organisms. Vancomycin was the most efficient against gram positive organisms.

In our study 30 patients had osteomyelitis, most of the patients had osteomyelitis of phalanx and metatarsals, most of the osteomyelitis of foot begin as chronic perforating ulcers. If the ulcer is ignored or inappropriately cared bacterial invasion may progress at the base of the lesion by spreading along the fascial planes or by perforating.

Neuropathy

Neuropathy foot has got three main complications neuropathic ulcer, neuropathic joint (Charcot's) and neuropathic edema.

Neuropathy consists of three main components – sensory, motor and autonomic.

In our series 52% patients had neuropathy. It was diagnosed by weakness of small muscles, foot deformity, loss of sensation, loss of sweating, formation of new pressure points, callosities and eventually ulcer.

In autonomic neuropathy skin becomes dry as a result of loss of sweating. There will be cracking of skin and impaired defense against infection. In case of sensory neuropathy there will be loss of sensation which predispose to the trauma and ulcer formation.

SPREAD OF INFECTION

After trauma there will be devitilisation of the tissue, if the infection supervenes there is rapid formation of inflammatory exudates, especially in the deeper underlying tissue which leads increased pressure in the compartment resulting in obstruction to the blood supply, eventually resulting in gangrene and uncontrolled infection.

Due to vasculopathy and neuropathy, foot infections are deserted, there is spread of the infection from distal to the proximal part.

Factors which are responsible for impaired wound healing in diabetes are

- Decreased vascularity
- Reduced resistance to infection
- Retained devitilised tissue due to vasculopathy and neuropathy
- Oedma due to inflammation
- Poor nourishment
- Repeated trauma

Co- relation of atherosclerosis and diabetic ulcers

As seen in various studies. Diabetes leads to microangiopathy and macroangiopathy with resultant atherosclerosis of blood vessels leading to decreased vascularity to the distal lower limb causing ulcerations, gangrenes etc.

Our study also corroborates these findings by showing high incidence and atherosclerosis in the patients with diabetic ulcers which can be considered as one of the causes.

DURATION OF HOSPITALIZATION

In this study minimum duration in hospital was 7 days (1week) and maximum was 84 days (12 weeks). Most of patients stay in hospital for 4-6 week.

This long duration of hospitalization can be explained by the refractory to patients diminished resistance of body due to uncontrolled sugar level, resistance of the organism to the antibiotic therapy, poor nutrition, advanced age.

INVESTIGATIONS

The study indicated that at the time of admission in hospital, RBS level of 70 patients were above the normal level and FBS level of patients done that showed 155 patients had more than normal range. It is recommended that diabetic patients present with diabetic foot lesions in uncontrollable blood sugar level. 75 patients showing normal blood sugar level because they are regularly taking anti-diabetic treatment.

TREATMENT

In our study diabetic foot management is a difficult task because of

- Illiteracy and ignorance
- Walking barefoot and poor socioeconomic status.
- Taking treatment in earlier stages by quacks because of fear of amputation.
- Careless removal of nails and unhygienic foot care.

Most of the cases in our study were referred to us in a difficult stage with serious infection and associated complications.

Before starting treatment every patient must be assessed to decide the relative importance of predisposing factors – neuropathy or vascular disease. In absence of severe ischemia, conservative medical and surgical management give good results. In presence of ischemia unless the limb is salvaged by direct arterial surgery, we may have to consider major amputations.

MEDICAL MANAGEMENT

For successful management it is important to assess the diabetic status, severity of infection and general nourishment of the patient.

Control of Diabetes mellitus by:-

- Diabetic diet

- Oral hypoglycemic drugs
- Insulin therapy

All our patients were advised diabetic diet with low cholesterol and high PUFA.

All patients were shifted to crystalline insulin therapy. According to the blood sugar level dosage of the insulin was adjusted. In case of Ketoacidosis, planned aggressive treatment was necessary because this is life threatening complication, where severity of infection is more and there is defective neutrophil function.

Principles of the treatment are:

- Correction of fluid and electrolyte imbalance
- Reversal of acidosis and ketosis with crystalline insulin therapy.

Dosage of insulin was required more in cases of ketoacidosis, severe infection. After correction of ketoacidosis and control of infection, the dosage of insulin required was reduced.

At the time of discharge 100 patients were shifted to oral hypoglycemic, 115 patients to Lente insulin and the rest were continued with the crystalline insulin.

Control of infection:-

Control of infection was done by careful debridement and antibiotic therapy. The role of antibiotics in foot infection is to limit the spread of infection.

Indications of antibiotic therapy were:-

- Limiting Cellilitis and spreading infection
- Prevent secondary infection
- Prophylactic therapy before surgery.

In our study most of the patients were treated with multidrug regime.

In our study we have used higher antibiotics because it is known to grow resistant organisms in diabetic foot infection.

Staphylococcus was the commonest organism grown, which was sensitive to Cefotaxime, Ampicillin and Gentamycin in 80-90% of cases.

We have used combination antibiotic therapy. Combination antibiotic therapy is advised by Brodsky J.W.et al & L.Bhasker Reddy et al .

Brodsky J.W. Advised the following combinations:-

-Ticarcillin + Clavulanic acid

-Vancomycin + Metronidazole

-Third generation cephalosporin + Metronidazole

L. Bhasker Reddy et al advised the following combination:-

Cefotaxime + Metronidazole

According to the availability of drugs we could use broad spectrum antibiotics like Ciprofloxacin, Cefotaxime, in combination with Metronidazole and Gentamycin.

Ampicillin, penicillin, erythromycin, tetracycline were used in some of the patients.

In the present series, 125 cases were treated by slough excision, 40 with skin graft, 35 by fasciotomy and I&D. Below Knee Amputation was done in 30 patients and Above Knee

Amputation were done in 10 cases. Minor amputations are done in 10 cases. Proper control of diabetes is very important in diabetic foot management. Fasting and PPBS estimations were well under control. Urine sugar estimation was done thrice daily.

Infection was treated with broad spectrum antibiotics. Patients were educated about care of foot and Pentoxyphylline was administered to in patients with ischemic lesions.

In our study amputation rate is 20%, that is lower when compared to standard studies. This could be due to better education of patient, better glycemic control, proper care of foot, proper use of antibiotics, extensive

debridement and regular dressing. After amputation, wound healed well. The patients were referred to rehabilitation center for prosthesis.

RESULTS

Out of 250 patients studied, 240 recovered and discharged from hospital and 10 patients died during the course of treatment due to various diabetes related complications like septicemia. Ketoacidosis, uremia and respiratory symptoms.

SUMMARY

Foot ulcers are one of the major complications of diabetes; they have a poor tendency to heal, which may result in long stay in hospital for treatment. The foot in diabetic patient is the cross road of several of several pathological processes, in which almost all components of lower extremity are involved- skin, subcutaneous tissue, muscles, bones, joints, blood vessels and nerves. An understanding of the development of the complications and application of preventive and management strategies will reduce the complications of diabetic foot. Patients who are diabetic should take proper care of their feet so that they will not suffer from the consequences. They should take proper medical care at the earliest once any trauma occurs, so that the chain of events could be halted.

With proper care by the attending surgeon which includes regular dressing, wound debridement, proper antibiotics the outcome can be favourable and avoid the unwanted surgeries like amputations.

CONCLUSION

This study comprised of 250 cases of diabetic foot patients with emphasis on surgical management and its complications. After analysis of the data the following are the conclusions.

The highest number of patients was seen in the age group of 51-60 years (32%). Males are almost two times more affected than females. Males are more at risk to trauma.

Farmers had more incidence of diabetic foot lesions.

Diabetic foot ulcers have seen more in NIDDM

Duration of diabetes varied from just diagnosed to 25 years.

In study 240 patients were known diabetic and 10 patients diagnosed at the time of presentation.

Insignificant trauma of some kind was the initiating factor in nearly one-third of the cases.

Minimum stay in hospital was 1 week and maximum 12 weeks, most of patients stay for 4-6 weeks.

Commonest presenting lesion was ulcer 48%, followed by Cellulitis 26% and gangrene 20%.

After 5 to 6 years of diabetes most of patients present with neuropathic lesions and they are in 35-80 years age group some of them develops gangrene.

Staphylococcus aureus is a commonest organism causing infection.

Atherosclerotic changes may lead to formation of ulceration of foot in diabetic patients.

Conservative treatment consisting of control of diabetes with plain / Lente insulin along with appropriate oral /IV antibiotics was effective in some cases.

Wound debridement, slough excision followed by dressing with Povidine iodine/ Magnesium sulphate/ Metrogyl/ Collagenase dressing resulted in healing in some cases. Split skin graft, disarticulations, Transmetatarsal amputation, Below Knee Amputation and Above Knee Amputation were other modes of treatment.

240 patients cured and 10 patients died during course of treatment.

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ANNEXURE

PROFORMA

Case No:-

Name:- Age:- Sex:- M/F

Occupation:-

IP No:- DOA:- DOD:-

Address:-

Socio economic status:-

Chief complaints:-

History of present illness:-

- Polyuria
- Polydypsia, Polyphagia
- Weakness
- Burning sensation of palms and sole
- Tingling /numbness
- Loss of appetite
- Loss of weight
- Ulceration
- Gangrene
- Neuropathy
- Poor wound healing

- Recurrent infections
- Fever
- Claudication
- Duration
- Distance
- Intermittent pain, Rest pain
- Any other associated history

Diabetic history:

- Type of diabetes:- NIDDM/IDDM
- Duration of Diabetes
- Controlled/ uncontrolled
- Duration of control and type of control

  -Good

  -Poor

  -Bad
- Method of treatment /control

  -Diet

  - Oral Hypoglycemic Agent: Dose

  - Insulin: Dose

  -Operative : Incision/Drainage/Excision/Amputation

Past history

- Visual disturbance
- Septic lesions - Ulcerations / Gangrene / Carbuncle / Furuncle / Abscess
- CVS - Angina / Myocardial infarction
- CNS: TIA / Peripheral neuropathy
- Recurrent Systemic infections
- H/O previous hospital admissions

  Number

  Duration

  Treatment

  Cured/ Not cured

Family history :- Family members suffering from DM- Y/N

Personal history:-

- Diet: Veg/Non veg
- Appetite: Normal/Increased/Decreased
- Micturition: Normal/Polyuria
- bowel: Regular/Irregular
- Smoker: Y/N
- Alcoholic consumption :Y/N
- History of prolong intake of drug: Y/N: Duration

Menstrual history:- in Females

GENERAL PHYSICAL EXAMINATION:-

- Built : Well / Moderate / Poor
- Nourishment: Well / Moderate / Poor
- Pallor / Icterus / Clubbing / Cyanosis / Oedema / Lymphadenopathy
- Vitals :-

Pulse: /min BP: RR: Temp:

SYSTEMIC EXAMINATION

Nervous system

Central:

Peripheral:

-Motor

- Sensory

- Reflexes

Respiratory system

Cardiovascular system

Abdomen examination

LOCAL EXAMINATION

- Site

Toe, Sole, Heel, Web space

- Other foot lesions- Dorsum/ Lateral borders
- Mode of presentation:

- Ulcer
- Cellulitis
- Blister
- Abscess
- Gangrene

- Local temperature
- Tenderness: Y/N
- Peripheral Neuropathy:- Y/N
- Sensation:- Touch / Vibration / Joint
- Skin/ Nail changes
- Presence/ Absence of callosities – Dry / Wet
- Peripherl Pulses:

  -Dorsalis pedis artery - Present / Weak / Absent

  - Posterior tibial artery – Present / Weak / Absent

INVESTIGATIONS

- Urine - Albumin / Sugar / Microscopy / Ketone bodies
- Blood – Hb% / Total count / Differential count
- FBS- PPBS-
- Blood urea: Serum Creatinine:
- Lipid profile
- Serum electrolytes
- X ray – Chest/Foot
- ECG
- Wound Swab for C&S

SPECIAL INVESTIGATIONS: Arterial Doppler

TREATMENT

- Diet
- Oral Hypoglycemic Agents: Dose
- Insulin: Dose
- Antibiotics
- Dressing : Glycerine Magnesium Sulphate/ Soframycin/ Betadine
- Supplementary treatment
- Preoperative treatment
- Anaesthesia used

- Surgical:-

- I&D
- Slough excision
- Fascitomy
- SSG
- Amputation / RA / TMA

- Post operative treatment
- Discharge notes
- Date of discharge
- Condition of patient
- Nature of operation
- Advise given

  Diet, Treatment, Change of Occupation, other advice.

  FOLLOW UP:- General condition/ local condition/ Complications/ Recurrence of fresh lesions.

ஒப்புதல் படிவம்

எனது சர்க்கரை நோயால் காலில் புண் இருப்பதை மருத்துவர் மூலம் அறிந்து கொண்டேன். அந்த சர்க்கரை நோய் புண்ணில் உள்ள கிருமியை பற்றி அறியவும் அதற்கான வைத்தியத்தை பற்றியும், அந்த புண்ணால் ஏற்படும் பாதிப்பு பற்றியும் மருத்துவர் விளக்கினார். அந்த புண்ணை பராமரிப்பு செய்வதை பற்றியும் மருத்துவர் விளக்கினார். அந்த புண்ணை சுத்தம் செய்வது பற்றியும் மருத்துவர் விளக்கினார். இவை அனைத்தை பற்றியும் ஆராய்ச்சி செய்வதை பற்றி மருத்துவர் விளக்கினார். இவை அனைத்தும் அறிந்து முழு மனதுடன் இதற்கு சம்மதம் அளிக்கிறேன்.

இப்படிக்கு

MASTER CHART

SL. NO	IP/OP NO	AGE	SEX	OCCUPATION	STAY IN HOSPITAL (DAYS)	TYPE OF DM	DURATION OF DM (YEARS)	MODE OF PRESENTATION	NEUROPATHY	ATHEROSCLEROTIC CHANGES	CULTURE	RBS	FBS	TREATMENT	OUTCOME
1	112871	43	M	Farmer	65	II	1	U	N	Y	S	N	N	D	Recover
2	22187	39	M	M L	23	II	2	U	N	N	P	>N	N	D	Recover
3	119181	53	F	M L	40	II	8	C	Y	Y	S	>N	>N	SE	Recover
4	133215	61	F	H W	38	II	5	U	Y	Y	K	N	N	SSG	Recover
5	144578	52	M	Business	14	II	10	A	N	N	S	>N	>N	I&D	Recover
6	146781	69	M	Farmer	46	II	14	U	Y	Y	Pr	>N	>N	D	Recover
7	29186	47	F	M L	28	II	9 Month	C	N	N	S	>N	>N	FASC	Recover
8	30897	33	F	Farmer	30	I	8	U	Y	Y	K	N	N	SSG	Recover
9	143571	53	M	Farmer	36	II	4	U	N	N	S	>N	>N	D	Recover
10	31059	63	M	Farmer	55	II	11	G	Y	Y	P	>N	>N	TMA	Recover
11	28958	73	M	Farmer	40	II	>20	U	N	N	E	N	N	SE	Recover
12	141098	43	M	O E	36	II	1	G	Y	N	S	>N	>N	RA	Recover
13	22154	64	F	H W	25	II	12	C	N	Y	K	>N	N	D	Death
14	140981	66	F	H W	20	II	>20	U	Y	N	S	>N	>N	D	Recover
15	143450	49	M	Farmer	28	II	Ne D	C	N	Y	NHS	>N	>N	D	Recover
16	31456	57	M	Farmer	62	II	9	U	Y	N	E	N	N	SSG	Recover
17	31876	69	M	Farmer	72	II	10	C	Y	Y	P	>N	>N	SE	Recover
18	31487	61	M	Farmer	27	II	10	G	Y	Y	K	>N	>N	AKA	Recover
19	30958	58	M	Business	12	II	8	U	Y	N	S	>N	N	D	Recover
20	138901	19	M	M L	32	I	1	U	N	N	P	N	N	D	Recover
21	39341	64	F	H W	46	II	8	G	Y	Y	Pr	>N	>N	BKA	Recover
22	131408	53	F	Farmer	42	II	11	U	N	Y	S	N	N	D	Recover
23	130938	44	M	Farmer	36	II	4	U	N	N	P	N	N	D	Recover
24	31786	65	M	Farmer	18	II	12	U	Y	Y	E	>N	>N	SE	Recover
25	31945	67	M	Farmer	29	II	18	C	Y	Y	S	N	N	FASC	Recover
26	36098	64	M	Farmer	52	II	16	U	Y	Y	E	>N	>N	SSG	Recover
27	35798	79	F	H W	25	II	19	A	Y	N	S	>N	>N	I&D	Recover
28	110958	42	M	Driver	22	I	3 Month	A	N	Y	Pr	>N	>N	I&D	Recover
29	101893	34	M	Farmer	7	I	9	U	N	Y	P	>N	N	D	Recover
30	33946	52	M	Farmer	36	II	Ne D	G	Y	Y	S	>N	>N	BKA	Recover
31	33914	75	F	H W	56	II	>20	U	N	N	K	>N	>N	SSG	Recover

32	33046	62	F	H W	42	II	15	C	Y	Y	S	N	N	SE	Recover
33	109815	41	F	Farmer	40	II	4	U	N	N	NHS	>N	>N	D	Recover
34	108956	55	M	Business	17	II	7	U	Y	Y	K	N	N	D	Recover
35	33785	66	M	Farmer	62	II	14	C	Y	Y	S	N	N	FASC	Recover
36	109089	63	F	H W	28	II	16	U	Y	Y	Pr	N	N	D	Recover
37	30186	59	F	Farmer	30	II	9	U	Y	Y	E	>N	>N	SSG	Recover
38	118764	44	M	M L	26	II	9 Month	C	N	Y	S	>N	N	D	Recover
39	34596	69	M	Business	48	II	14	U	Y	N	E	>N	>N	SSG	Recover
40	139085	84	M	Farmer	24	II	>20	U	Y	Y	K	N	N	D	Recover
41	133569	51	M	O E	19	II	8	A	N	N	S	>N	>N	I&D	Recover
42	38641	47	M	Driver	73	II	7	C	N	N	NHS	>N	>N	FASC	Recover
43	41509	66	M	Farmer	37	II	17	U	N	Y	S	>N	>N	SE	Recover
44	45096	22	F	Farmer	26	I	4	G	Y	Y	K	>N	>N	BKA	Recover
45	21258	53	M	Farmer	50	II	7	U	Y	Y	NHS	>N	>N	D	Death
46	34081	62	F	H W	38	II	19	U	N	Y	K	>N	>N	SSG	Recover
47	11096	46	F	Farmer	40	II	9Month	U	Y	Y	S	N	N	SE	Recover
48	131809	54	F	Business	52	II	8	U	N	Y	P	>N	>N	D	Recover
49	131560	35	M	Farmer	34	II	5	C	Y	Y	E	N	N	D	Recover
50	35085	58	M	Farmer	38	II	8	G	Y	Y	Pr	>N	>N	AKA	Recover
51	45096	67	M	Farmer	20	II	17	U	N	Y	S	>N	>N	SSG	Recover
52	136801	43	M	Farmer	40	II	7Month	C	N	N	P	N	N	D	Recover
53	48174	54	M	Farmer	60	II	6	C	Y	Y	K	>N	N	FASC	Recover
54	31267	62	M	Farmer	28	II	13	U	Y	Y	S	>N	>N	BKA	Death
55	136578	50	F	M L	13	II	7	G	Y	Y	E	>N	>N	D	Recover
56	139083	32	F	Farmer	37	I	9	U	Y	Y	S	N	N	D	Recover
57	136045	33	M	Farmer	45	I	8	C	Y	N	S	N	N	D	Recover
58	19817	59	M	Farmer	80	II	10	U	Y	Y	K	>N	N	SSG	Recover
59	15908	60	M	Farmer	26	II	1	G	Y	Y	S	>N	>N	SE	Recover
60	34843	60	M	Farmer	24	II	10	G	Y	Y	Pr	>N	>N	BKA	Recover
61	34957	76	F	Business	12	II	6	C	N	N	S	N	N	SSG	Recover
62	134768	41	M	H W	42	II	>20	U	Y	N	E	>N	>N	D	Recover
63	134096	57	F	Farmer	56	II	4	G	Y	Y	P	>N	>N	D	Recover
64	35793	42	F	H W	23	II	8Month	G	Y	Y	S	>N	>N	BKA	Recover
65	38093	62	M	M L	28	I	9	U	N	Y	K	>N	>N	SE	Recover
66	136098	54	M	Farmer	30	II	16	C	N	N	E	N	N	D	Recover
67	10281	49	M	Farmer	32	II	11	U	Y	Y	S	>N	>N	D	Death
68	21198	53	M	Driver	36	II	5	G	Y	Y	E	>N	>N	BKA	Recover
69	111903	61	F	H W	60	II	6	U	N	Y	S	N	N	SE	Recover
70	110895	28	F	H W	40	II	15	U	N	Y	S	>N	>N	D	Recover
71	101095	52	M	Farmer	34	I	12	U	N	N	S	>N	>N	D	Recover

72	119807	59	F	Business	16	II	6	G	Y	Y	K	>N	>N	TMA	Recover
73	104575	37	M	Farmer	36	II	10	U	N	Y	S	>N	>N	S	Recover
74	36098	60	F	Farmer	30	II	1	G	Y	Y	Pr	>N	>N	BKA	Recover
75	101834	67	M	Business	32	II	16	U	N	Y	P	N	N	D	Recover
76	146786	45	M	Farmer	84	II	>20	U	Y	N	S	>N	>N	D	Recover
77	134876	51	F	Farmer	40	II	6	C	N	N	NHS	>N	N	SE	Recover
78	31908	66	M	H W	17	II	5	G	Y	N	S	>N	>N	BKA	Recover
79	34675	48	M	Farmer	30	II	18	U	N	Y	K	N	N	SSG	Recover
80	156093	55	M	M L	48	II	4Month	C	N	Y	S	>N	>N	D	Recover
81	109814	59	F	Farmer	22	II	9	A	N	N	K	>N	>N	I&D	Recover
82	118905	43	F	H W	24	I	14	U	Y	Y	S	>N	>N	D	Recover
83	46186	60	M	Business	30	II	19	U	N	Y	E	N	N	D	Recover
84	45098	39	F	Farmer	11	I	3	C	N	N	S	N	N	FASC	Recover
85	54190	32	M	H W	28	I	15	G	Y	Y	P	>N	>N	AKA	Recover
86	55409	51	M	Driver	62	II	1	U	N	Y	K	N	N	SSG	Recover
87	54318	42	F	Farmer	36	II	11	G	Y	Y	S	>N	>N	BKA	Recover
88	146789	72	M	H W	32	II	7	U	Y	Y	Pr	>N	>N	D	Recover
89	60189	58	F	Farmer	26	II	>20	G	Y	Y	S	>N	>N	TPA	Recover
90	176589	64	M	H W	48	II	Ne D	U	N	N	K	N	N	SE	Recover
91	146036	38	F	Farmer	37	II	9	C	N	Y	P	>N	>N	D	Recover
92	141809	57	M	O E	32	II	2	U	N	N	S	>N	>N	D	Recover
93	16617	46	M	Farmer	39	II	18	C	N	N	Pr	>N	>N	FASC	Recover
94	146087	63	F	Business	15	II	9Month	C	N	Y	S	>N	>N	D	Recover
95	18908	55	M	H W	40	II	10	U	N	N	E	N	N	SSG	Recover
96	19080	42	M	Farmer	32	I	2	G	Y	Y	NHS	>N	>N	BKA	Recover
97	54190	67	F	Farmer	24	I	15	A	N	N	K	>N	>N	I&D	Recover
98	19015	60	M	H W	26	II	>20	C	N	N	S	N	N	S	Recover
99	145068	60	M	Farmer	70	II	9	C	N	N	E	>N	>N	D	Recover
100	14576	78	M	Farmer	44	II	19	G	Y	N	Pr	>N	>N	BKA	Recover
101	143164	43	M	Farmer	65	II	1	U	N	Y	S	N	N	D	Recover
102	140183	39	M	M L	23	II	2	U	N	N	P	>N	N	D	Recover
103	35906	53	F	M L	40	II	8	C	Y	Y	S	>N	>N	SE	Recover
104	22906	61	F	H W	38	II	5	U	Y	Y	K	N	N	SSG	Recover
105	135068	52	M	Business	14	II	10	A	N	N	S	>N	>N	I&D	Recover
106	13906	69	M	Farmer	46	II	14	U	Y	Y	Pr	>N	>N	D	Recover
107	41807	47	F	M L	28	II	9 Month	C	N	N	S	>N	>N	FASC	Recover
108	31765	33	F	Farmer	30	I	8	U	Y	Y	K	N	N	SSG	Recover
109	138041	53	M	Farmer	36	II	4	U	N	N	S	>N	>N	D	Recover
110	19199	63	M	Farmer	55	II	11	G	Y	Y	P	>N	>N	TMA	Recover
111	110065	73	M	Farmer	40	II	>20	U	N	N	E	N	N	SE	Recover

112	19931	43	M	O E	36	II	1	G	Y	N	S	>N	>N	RA	Recover
113	10985	64	F	H W	25	II	12	C	N	Y	K	>N	N	D	Death
114	110083	66	F	H W	20	II	>20	U	Y	N	S	>N	>N	D	Recover
115	104583	49	M	Farmer	28	II	Ne D	C	N	Y	NHS	>N	>N	D	Recover
116	41586	57	M	Farmer	62	II	9	U	Y	N	E	N	N	SSG	Recover
117	45386	69	M	Farmer	72	II	10	C	Y	Y	P	>N	>N	SE	Recover
118	47809	61	M	Farmer	27	II	10	G	Y	Y	K	>N	>N	AKA	Recover
119	110981	58	M	Business	12	II	8	U	Y	N	S	>N	N	D	Recover
120	143156	19	M	M L	32	I	1	U	N	N	P	N	N	D	Recover
121	19803	64	F	H W	46	II	8	G	Y	Y	Pr	>N	>N	BKA	Recover
122	178601	53	F	Farmer	42	II	11	U	N	Y	S	N	N	D	Recover
123	177098	44	M	Farmer	36	II	4	U	N	N	P	N	N	D	Recover
124	18096	65	M	Farmer	18	II	12	U	Y	Y	E	>N	>N	SE	Recover
125	22096	67	M	Farmer	29	II	18	C	Y	Y	S	N	N	FASC	Recover
126	22305	64	M	Farmer	52	II	16	U	Y	Y	E	>N	>N	SSG	Recover
127	100186	79	F	H W	25	II	19	A	Y	N	S	>N	>N	I&D	Recover
128	100786	42	M	Driver	22	I	3 Month	A	N	Y	Pr	>N	>N	I&D	Recover
129	103147	34	M	Farmer	7	I	9	U	N	Y	P	>N	N	D	Recover
130	15698	52	M	Farmer	36	II	Ne D	G	Y	Y	S	>N	>N	BKA	Recover
131	14908	75	F	H W	56	II	>20	U	N	N	K	>N	>N	SSG	Recover
132	16098	62	F	H W	42	II	15	C	Y	Y	S	N	N	SE	Recover
133	130981	41	F	Farmer	40	II	4	U	N	N	NHS	>N	>N	D	Recover
134	141096	55	M	Business	17	II	7	U	Y	Y	K	N	N	D	Recover
135	18073	66	M	Farmer	62	II	14	C	Y	Y	S	N	N	FASC	Recover
136	141096	63	F	H W	28	II	16	U	Y	Y	Pr	N	N	D	Recover
137	17068	59	F	Farmer	30	II	9	U	Y	Y	E	>N	>N	SSG	Recover
138	140387	44	M	M L	26	II	9 Month	C	N	Y	S	>N	N	D	Recover
139	17809	69	M	Business	48	II	14	U	Y	N	E	>N	>N	SSG	Recover
140	110981	84	M	Farmer	24	II	>20	U	Y	Y	K	N	N	D	Recover
141	17065	51	M	O E	19	II	8	A	N	N	S	>N	>N	I&D	Recover
142	17408	47	M	Driver	73	II	7	C	N	N	NHS	>N	>N	FASC	Recover
143	17046	66	M	Farmer	37	II	17	U	N	Y	S	>N	>N	SE	Recover
144	17487	22	F	Farmer	26	I	4	G	Y	Y	K	>N	>N	BKA	Recover
145	11358	53	M	Farmer	50	II	7	U	Y	Y	NHS	>N	>N	D	Death
146	18065	62	F	H W	38	II	19	U	N	Y	K	>N	>N	SSG	Recover
147	11567	46	F	Farmer	40	II	9Month	U	Y	Y	S	N	N	SE	Recover
148	110080	54	F	Business	52	II	8	U	N	Y	P	>N	>N	D	Recover
149	110908	35	M	Farmer	34	II	5	C	Y	Y	E	N	N	D	Recover
150	31341	58	M	Farmer	38	II	8	G	Y	Y	Pr	>N	>N	AKA	Recover
151	34765	67	M	Farmer	20	II	17	U	N	Y	S	>N	>N	SSG	Recover

152	43187	43	M	Farmer	40	II	7Month	C	N	N	P	N	N	D	Recover
153	18046	54	M	Farmer	60	II	6	C	Y	Y	K	>N	N	FASC	Recover
154	13576	62	M	Farmer	28	II	13	U	Y	Y	S	>N	>N	BKA	Death
155	144096	50	F	M L	13	II	7	G	Y	Y	E	>N	>N	D	Recover
156	180916	32	F	Farmer	37	I	9	U	Y	Y	S	N	N	D	Recover
157	156781	33	M	Farmer	45	I	8	C	Y	N	S	N	N	D	Recover
158	15890	59	M	Farmer	80	II	10	U	Y	Y	K	>N	N	SSG	Recover
159	14509	60	M	Farmer	26	II	1	G	Y	Y	S	>N	>N	SE	Recover
160	13099	60	M	Farmer	24	II	10	G	Y	Y	Pr	>N	>N	BKA	Recover
161	18098	76	F	Business	12	II	6	C	N	N	S	N	N	SSG	Recover
162	154196	41	M	H W	42	II	>20	U	Y	N	E	>N	>N	D	Recover
163	155091	57	F	Farmer	56	II	4	G	Y	Y	P	>N	>N	D	Recover
164	18801	42	F	H W	23	II	8Month	G	Y	Y	S	>N	>N	BKA	Recover
165	15544	62	M	M L	28	I	9	U	N	Y	K	>N	>N	SE	Recover
166	155578	54	M	Farmer	30	II	16	C	N	N	E	N	N	D	Recover
167	17854	49	M	Farmer	32	II	11	U	Y	Y	S	>N	>N	D	Death
168	11096	53	M	Driver	36	II	5	G	Y	Y	E	>N	>N	BKA	Recover
169	14851	61	F	H W	60	II	6	U	N	Y	S	N	N	SE	Recover
170	154187	28	F	H W	40	II	15	U	N	Y	S	>N	>N	D	Recover
171	151308	52	M	Farmer	34	I	12	U	N	N	S	>N	>N	D	Recover
172	17303	59	F	Business	16	II	6	G	Y	Y	K	>N	>N	TMA	Recover
173	18011	37	M	Farmer	36	II	10	U	N	Y	S	>N	>N	S	Recover
174	19901	60	F	Farmer	30	II	1	G	Y	Y	Pr	>N	>N	BKA	Recover
175	153409	67	M	Business	32	II	16	U	N	Y	P	N	N	D	Recover
176	159081	45	M	Farmer	84	II	>20	U	Y	N	S	>N	>N	D	Recover
177	15081	51	F	Farmer	40	II	6	C	N	N	NHS	>N	N	SE	Recover
178	14083	66	M	H W	17	II	5	G	Y	N	S	>N	>N	BKA	Recover
179	15305	48	M	Farmer	30	II	18	U	N	Y	K	N	N	SSG	Recover
180	157809	55	M	M L	48	II	4Month	C	N	Y	S	>N	>N	D	Recover
181	151605	59	F	Farmer	22	II	9	A	N	N	K	>N	>N	I&D	Recover
182	18756	43	F	H W	24	I	14	U	Y	Y	S	>N	>N	D	Recover
183	189017	60	M	Business	30	II	19	U	N	Y	E	N	N	D	Recover
184	19435	39	F	Farmer	11	I	3	C	N	N	S	N	N	FASC	Recover
185	19683	32	M	H W	28	I	15	G	Y	Y	P	>N	>N	AKA	Recover
186	17058	51	M	Driver	62	II	1	U	N	Y	K	N	N	SSG	Recover
187	15078	42	F	Farmer	36	II	11	G	Y	Y	S	>N	>N	BKA	Recover
188	180710	72	M	H W	32	II	7	U	Y	Y	Pr	>N	>N	D	Recover
189	37865	58	F	Farmer	26	II	>20	G	Y	Y	S	>N	>N	TMA	Recover
190	109873	64	M	H W	48	II	Ne D	U	N	N	K	N	N	SE	Recover
191	108786	38	F	Farmer	37	II	9	C	N	Y	P	>N	>N	D	Recover

192	109457	57	M	O E	32	II	2	U	N	N	S	>N	>N	D	Recover
193	37890	46	M	Farmer	39	II	18	C	N	N	Pr	>N	>N	FASC	Recover
194	106793	63	F	Business	15	II	9Month	C	N	Y	S	>N	>N	D	Recover
195	37908	55	M	H W	40	II	10	U	N	N	E	N	N	SSG	Recover
196	37935	42	M	Farmer	32	I	2	G	Y	Y	NHS	>N	>N	BKA	Recover
197	108954	67	F	Farmer	24	I	15	A	N	N	K	>N	>N	I&D	Recover
198	110954	60	M	H W	26	II	>20	C	N	N	S	N	N	S	Recover
199	107434	60	M	Farmer	70	II	9	C	N	N	E	>N	>N	D	Recover
200	37978	78	M	Farmer	44	II	19	G	Y	N	Pr	>N	>N	BKA	Recover
201	108573	43	M	Farmer	65	II	1	U	N	Y	S	N	N	D	Recover
202	105793	39	M	M L	23	II	2	U	N	N	P	>N	N	D	Recover
203	104809	53	F	M L	40	II	8	C	Y	Y	S	>N	>N	SE	Recover
204	100393	61	F	H W	38	II	5	U	Y	Y	K	N	N	SSG	Recover
205	37987	52	M	Business	14	II	10	A	N	N	S	>N	>N	I&D	Recover
206	93476	69	M	Farmer	46	II	14	U	Y	Y	Pr	>N	>N	D	Recover
207	37998	47	F	M L	28	II	9 Month	C	N	N	S	>N	>N	FASC	Recover
208	38008	33	F	Farmer	30	I	8	U	Y	Y	K	N	N	SSG	Recover
209	90854	53	M	Farmer	36	II	4	U	N	N	S	>N	>N	D	Recover
210	38045	63	M	Farmer	55	II	11	G	Y	Y	P	>N	>N	TMA	Recover
211	104876	73	M	Farmer	40	II	>20	U	N	N	E	N	N	SE	Recover
212	38101	43	M	O E	36	II	1	G	Y	N	S	>N	>N	RA	Recover
213	19182	64	F	H W	25	II	12	C	N	Y	K	>N	N	D	Death
214	98765	66	F	H W	20	II	>20	U	Y	N	S	>N	>N	D	Recover
215	113546	49	M	Farmer	28	II	Ne D	C	N	Y	NHS	>N	>N	D	Recover
216	38931	57	M	Farmer	62	II	9	U	Y	N	E	N	N	SSG	Recover
217	113765	69	M	Farmer	72	II	10	C	Y	Y	P	>N	>N	SE	Recover
218	38945	61	M	Farmer	27	II	10	G	Y	Y	K	>N	>N	AKA	Recover
219	119678	58	M	Business	12	II	8	U	Y	N	S	>N	N	D	Recover
220	143789	19	M	M L	32	I	1	U	N	N	P	N	N	D	Recover
221	38654	64	F	H W	46	II	8	G	Y	Y	Pr	>N	>N	BKA	Recover
222	133098	53	F	Farmer	42	II	11	U	N	Y	S	N	N	D	Recover
223	109876	44	M	Farmer	36	II	4	U	N	N	P	N	N	D	Recover
224	138765	65	M	Farmer	18	II	12	U	Y	Y	E	>N	>N	SE	Recover
225	38765	67	M	Farmer	29	II	18	C	Y	Y	S	N	N	FASC	Recover
226	38674	64	M	Farmer	52	II	16	U	Y	Y	E	>N	>N	SSG	Recover
227	119087	79	F	H W	25	II	19	A	Y	N	S	>N	>N	I&D	Recover
228	101001	42	M	Driver	22	I	3 Month	A	N	Y	Pr	>N	>N	I&D	Recover
229	109845	34	M	Farmer	7	I	9	U	N	Y	P	>N	N	D	Recover
230	38706	52	M	Farmer	36	II	Ne D	G	Y	Y	S	>N	>N	BKA	Recover
231	38810	75	F	H W	56	II	>20	U	N	N	K	>N	>N	SSG	Recover

232	111987	62	F	H W	42	II	15	C	Y	Y	S	N	N	SE	Recover
233	135876	41	F	Farmer	40	II	4	U	N	N	NHS	>N	>N	D	Recover
234	131864	55	M	Business	17	II	7	U	Y	Y	K	N	N	D	Recover
235	38901	66	M	Farmer	62	II	14	C	Y	Y	S	N	N	FASC	Recover
236	104576	63	F	H W	28	II	16	U	Y	Y	Pr	N	N	D	Recover
237	38911	59	F	Farmer	30	II	9	U	Y	Y	E	>N	>N	SSG	Recover
238	118976	44	M	M L	26	II	9 Month	C	N	Y	S	>N	N	D	Recover
239	167851	69	M	Business	48	II	14	U	Y	N	E	>N	>N	SSG	Recover
240	190186	84	M	Farmer	24	II	>20	U	Y	Y	K	N	N	D	Recover
241	135436	51	M	O E	19	II	8	A	N	N	S	>N	>N	I&D	Recover
242	190690	47	M	Driver	73	II	7	C	N	N	NHS	>N	>N	FASC	Recover
243	110987	66	M	Farmer	37	II	17	U	N	Y	S	>N	>N	SE	Recover
244	38976	22	F	Farmer	26	I	4	G	Y	Y	K	>N	>N	BKA	Recover
245	21394	53	M	Farmer	50	II	7	U	Y	Y	NHS	>N	>N	D	Death
246	39786	62	F	H W	38	II	19	U	N	Y	K	>N	>N	SSG	Recover
247	117654	46	F	Farmer	40	II	9Month	U	Y	Y	S	N	N	SE	Recover
248	39891	54	F	Business	52	II	8	U	N	Y	P	>N	>N	D	Recover
249	39899	35	M	Farmer	34	II	5	C	Y	Y	E	N	N	D	Recover
250	40181	58	M	Farmer	38	II	8	G	Y	Y	Pr	>N	>N	AKA	Recover

ABBREVIATIONS

Ne D – Newly Diagnosed

S- Staphylococcus aureus

NHS- Non- Hemolytic Streptococci

P- Pseudomonas

K-Klebsiella

E- E.coli

Pr- Proteus

U- Ulcer

A- Abscess

G- Gangrene

C- Cellulitis

D- Debridement

SE- Slough Excision

SSG- Split Skin Grafting

I&D- Incision & Drainage

FASC- Fasciotomy

AKA- Above Knee Amputation

BKA- Below Knee Amputation

TMA- Trans- Metarsal Amputation

RA- Ray Amputation

O E- Office Employee

H W- Housewife

M L- Manual Labour